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QUANTITATIVE WASSERMANN TESTS IN DIAGNOSIS OF CONGENITAL SYPHILIS

CLINICAL IMPORTANCE OF FILDES' LAW

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The problem of deciding whether the infant of a syphilitic mother is also syphilitic and requires specific treatment is frequently difficult, particularly when the mother had been treated during her pregnancy and the baby shows no clinical manifestations of the disease but does have a positive serologic reaction in the blood from the cord or in the peripheral blood during the neonatal period. Fildes [1] in 1915, after studying 1,015 infants in a clinic in East London with special attenion to the question of syphilis, concluded: "The Wassermann reaction obtained with blood from the placental end of the cord is not diagnostic of syphilis in the infant but of syphilis in the mother." He further stated: "The phenomenon does not depend on the use of blood from the umbilical cord but is also met with when the blood is obtained direct from the infant." Fildes' observation that initially positive Wassermann reactions in infants frequently become negative without treatment and that the infants are not actually infected has been confirmed by Cruickshank [2] and others, most recently and convincingly by Dunham.[3] It is nevertheless common practice to consider a positive Wassermann reaction in a new-born infant as presumptive evidence of syphilis and to institute treatment on that basis alone. Obviously, such practice submits the infant to the not inconsiderable risks of arsphenamine therapy and

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1. Fildes, Paul: The Prevalence of Congenital Syphilis Amongst the Newly Born of the East End of London, J. Obst. & Gynaec. Brit. Emp. **27**:124, 1915.

2. Cruickshank, J. W.: Maternal Syphilis as a Cause of Death of the Foetus and of the New-Born Child: A Study of Congenital Syphilis Based on Serological, Clinical, and Post-Mortem Findings, Child Life Investigations, Medical Research Council, Special Report Series, no. 82, London, His Majesty's Stationery Office, 1924.

3. Dunham, E. C.: The Diagnosis of Congenital Syphilis in the New-Born, Am. J. Dis. Child. **43**:317 (Feb.) 1932.

also tends to falsify statistics on the curability of congenital syphilis. We agree emphatically with Moore's [4] opinion that "treatment should be withheld until the diagnosis of syphilis is definitely established . . . in the child, as in the adult, antisyphilitic treatment is not without risk, and it is a grave matter to enter into a long course of treatment in a child who may not have syphilis."

The proportion of cases in which a syphilitic mother transmits reagin to her infant, producing a temporarily positive Wassermann reaction without transmitting the disease itself is difficult to determine and probably varies in different localities and with different conditions of race and hygiene. It is general experience, however, that thorough treatment of mothers during pregnancy commonly prevents infection of the offspring, even though the serologic reaction of the mother herself remains positive. In such cases the serologic reaction of the infant is often also positive but, whether the child is treated or not, soon becomes negative. If he is treated, a misleading conception of the ease of cure will be obtained. We may cite the following observations of our own. Ten consecutive infants who were born of syphilitic mothers and whose blood gave a positive Wassermann reaction were given from one to three courses of antisyphilitic therapy, and all became normal within periods varying from one week to two months. They never showed any manifestations of syphilis, and at the age of 1 year the reaction of the spinal fluid in all cases was completely negative. The next ten similar infants were given no treatment at all, and all, likewise, became normal in respect to the reaction of the blood and spinal fluid and never showed clinical evidence of syphilis. These infants were from our own clinics, and their mothers had been treated during pregnancy. From the shortness of the period of treatment and the freedom from symptoms, as well as from comparison with the second series, it is fairly clear that the first series of infants had not—contrary to our belief at that time—been actually syphilitic.

Early differentiation of the syphilitic from the nonsyphilitic infant may, then, be a matter of great difficulty when clinical manifestations are lacking. Roentgenography is helpful, but it is costly, does not always show definitive changes early and requires interpretation by an experienced observer. It occurred to us that serial quantitative Wassermann tests might be of considerable value. For this purpose, we have followed Moore's plan of estimating the number of units of Wassermann reagin in the serum.

It cannot be too strongly emphasized that the routine Wassermann test is qualitative and has little quantitative value. The expression

4. Moore, J. E.: The Modern Treatment of Syphilis, Springfield, Ill., Charles C. Thomas, Publisher, 1933, p. 259.

"strongly positive" means little. Moore [5] said: "The Wassermann may be positive with minute amounts of serum, i. e., as little as .000125 cc. The routine report, however, will not disclose this fact; it will be only positive, 4 plus, or whatever other term is used to convey the fact of positivity, equally with the report of the patient whose serum contains only one one-hundredth as much reagin." Only by titration can the degree of positiveness be measured.

Our hypothesis in using the quantitative method was that with an initially positive reaction to a routine Wassermann test in an infant at or near the time of birth successive quantitative tests might reveal a progressive decline in titer to zero if syphilis were not present and the decline would be detectable considerably sooner than any change in the results of the ordinary routine test would be evident. If, on the other hand, the child were actually syphilitic the titer would not decline and might rise. If clinical signs of syphilis were absent and the titer was found to decline, treatment could be safely withheld, pending further observation.

METHOD

The ordinary technic for the Wassermann test, with cholesterolized antigen, was followed, only the amount of serum from the patient being varied. This was usually diluted 1:20 with physiologic solution of sodium chloride. In the standard test in the laboratory of the Stanford University School of Medicine 0.1 cc. of serum is used. For preliminary quantitative determination, a series of four tests, in addition to the standard test, using, respectively, 0.02 cc., 0.01 cc., 0.002 cc. and 0.001 cc., is suggested to show the probable range of positive reaction; intermediate or smaller amounts can then be used if further or more precise measurements are needed.

The unit [6] which we have found convenient may be expressed as the amount of reagin that in 0.1 cc. of serum is just sufficient to give a positive Wassermann reaction. The number of units in a given specimen equals one-tenth the reciprocal of the smallest amount of serum producing a positive reaction and can be easily computed or read directly from a slide rule. Thus, 0.1 cc. of serum is equivalent to 1 unit, 0.02 cc. to 5 units, 0.01 cc. to 10 units, 0.002 cc. to 50 units and 0.001 cc. to 100 units. The limits of precision depend mainly on the intervals between the amounts of serum used in the serial tests. Thus, if two successive tubes contain 0.02 and 0.01 cc. of serum and the reaction of only the first is positive, the actual amount of reagin lies between 5 and 9 units. When the result of the test reads $++-$, the unitage is probably almost exactly at the level corresponding with the quantity of serum employed.

Our quantitative tests were all made in the Wassermann laboratory of the Stanford University School of Medicine, with the cooperation of its head, Dr.

5. Moore,[4] p. 423.

6. This unit is apparently of different magnitude from Moore's. From his description we cannot make a definite comparison, but our impression is that ours is five times his.

H. A. Wyckoff. Some of the routine tests were made at the laboratory of the San Francisco Board of Health, with essentially the same technic as that used in the Stanford University laboratory.

The amounts of serum used in the cases to be reported differed somewhat from those we have suggested for preliminary titration; the latter will, however, we believe, be found convenient for routine work.

REPORT OF TEN CASES

CASES 1 and 2.—L. B., a boy, and C. B., a girl, were two ovum Negro twins, born on Nov. 22, 1934. The mother had been thoroughly treated for the last year or two, and her Wassermann reaction was said to have been negative during the pregnancy. Neither baby showed any evidence of disease.

TABLE 1.—*Results of Quantitative Wassermann Tests (Case 1)*

Date	Age	Amount of Serum, Cc.	Reaction	Units of Reagin
Nov. 27, 1934	5 days	0.1	+ + −	1
December 10	18 days	0.1	+ − −	
		0.08	− − −	0 +
January 16	7 weeks	0.1	− − −	0
April 24	5 months	0.1	− − −	0

TABLE 2.—*Results of Quantitative Wassermann Tests (Case 2)*

Date	Age	Amount of Serum, Cc.	Reaction	Units of Reagin
Nov. 22, 1934	Birth	0.1 (blood from the cord)	+ + + (B.H.*)	Not less than 1
November 27	5 days	0.1	+ + + (Stanford † Laboratory)	Not less than 1
December 10	18 days	0.1	+ − −	
		0.08	− − −	0 +
February 1	70 days	0.1	− − − (B.H.)	0

* B.H. indicates that the specimen was tested at the laboratories of the San Francisco Board of Health.

† Cholesterolized antigen was used; with acetone-insoluble antigen the reaction was + ± −; with alcoholic extract it was − − −.

L. B. showed no sign of syphilis at any time. The results of Wassermann tests are given in table 1.

On April 22 (age 5 months) the spinal fluid contained no cells; the reactions to the Nonne and Noguchi tests were negative; the Wassermann reaction was negative, and there was a colloidal gold curve of 1111000000.

C. B. showed no sign of syphilis; she had mild eczema at 5 months. The reaction to Wassermann tests are shown in table 2.

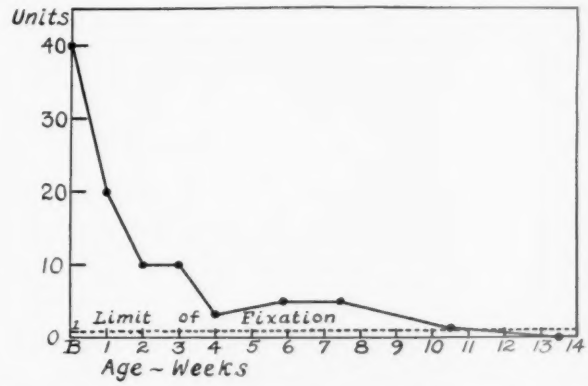
On April 24 (age 5 months) the Wassermann reaction of the spinal fluid was negative; there were no cells; the reactions to the Nonne and Noguchi tests were negative, and the colloidal gold curve was 1111100000.

CASE 3.—J. B. was a girl, born on Dec. 4, 1934, at full term; the weight at birth was 2,820 Gm. The mother stated that her mother and father had syphilis; she herself was said to have been syphilitic at birth but she did not receive treat-

ment until she was 12 years old. In September 1934, in the "prenatal clinic," her Wassermann reaction was +++, and since then she had been thoroughly treated (for the last six months of pregnancy). She had no clinical evidence of syphilis and no history of distinctive lesions. The baby, though small, showed no evidence of disease, except that at the age of 15 days there was a small "macerated" area of excoriation on the perineum; this healed without specific treatment. She was partly breast fed for a short time only. Growth was satisfactory. Roentgenograms were made on Jan. 14, 1935 (age 6 weeks), and the bones of the arms and legs showed no evidence of syphilis. The results of Wassermann tests are shown in table 3 and in the chart.

On May 15 the spinal fluid showed 1.1 leukocytes per cubic millimeter. The reactions to the Nonne and Noguchi tests were negative; the Wassermann reaction was negative, and the colloidal gold curve was 1111100000.

CASE 4.—E. B. M., a girl, was born on Jan. 7, 1935, at full term; the weight at birth was 9 pounds and 4 ounces (4,190 Gm.). The mother was found during pregnancy (8 weeks before delivery) to have a positive Wassermann reaction



The graph showing the progressive decline in the amount of reagin in the blood of the patient in case 3, without treatment. The mother herself had congenital syphilis and was treated thoroughly during her pregnancy. Her Wassermann reaction, however, remained positive. The serum of the cord blood contained 40 units of reagin per one-tenth cubic centimeter. The infant was given no antisyphilitic treatment. Note the sharp decline to 20 units on the seventh day and the more gradual subsequent decline. The line does not reach the point (limit of fixation) at which the reaction to the routine Wassermann test begins to weaken until the seventy-third day and does not reach zero until the ninety-fourth. The slight rise in the curve from the fourth to the sixth week is within the limits of accuracy of the quantitative test and has no significance.

and was given seven treatments with arsphenamine. The infection was probably recent, since her Wassermann reaction in a previous pregnancy was said to have been negative, and the child, who was 19 months old, had a negative Wassermann reaction. The younger child had no sign of disease. The reactions to the Wassermann tests are shown in table 4.

On April 12 (age 3 months and 7 days) the Wassermann reaction of the spinal fluid was negative; there were 2.2 leukocytes per cubic millimeter; the reactions to the Nonne and Noguchi tests were negative, and the colloidal gold curve was 1111100000. A Wassermann test was made on February 27 on the mother's milk, and the reaction was negative.

CASE 5.—C. W. H. was a boy, born on Jan. 13, 1935, at full term. The weight at birth was about 10 pounds (4,530 Gm.). The mother had syphilis about 1929.

TABLE 3.—*Results of Quantitative Wassermann Tests (Case 3)*

Date	Age	Amount of Serum, Cc.	Reaction	Units of Reagin
Dec. 4, 1934	Birth (Blood from cord)	0.005	+ + +	
		0.0025	+ + —	
		0.00175	— — —	40
December 11	7 days	0.005	+ + +	
		0.0025	— — —	20
December 19	15 days	0.01	+ + +	
		0.005	— — —	10
December 24	20 days	0.01	+ + +	
		0.005	— — —	10
Jan. 1, 1935	28 days	0.03	+ + +	
		0.01	— — —	3.3
January 9	36 days	0.03	+ + +	
		0.01	— — —	3.3
January 14	41 days	0.02	+ + +	
		0.01	— — —	5*
January 25	52 days	(Reaction was essentially the same as that on January 14)		
February 15	73 days	0.1	+ + —	
		0.07	+ + —	
		0.05	— — —	1 to 1.4
March 8	94 days	0.1	— — —	
		0.07	— — —	0
May 15	5½ months	0.1	— — — (B.H.)	

* This amount (0.02 cc.) of serum had not been used in the previous tests, when the interval was from 0.03 to 0.01. The 5 units of this test, therefore, cannot be interpreted as signifying an actual increase in the reagin content.

TABLE 4.—*Results of Quantitative Wassermann Tests (Case 4)*

Date	Age	Amount of Serum, Cc.	Reaction	Units of Reagin
Jan. 7, 1935	Birth	(Blood from cord)	+ + + + (B.H.)	
January 14	7 days	0.05	+ + +	
		0.03	— — —	2
February 26	50 days	0.01	— — —	
		0.07	— — —	0

She had a syphilitic baby about that year, who died at the age of 5 months, and she had had two miscarriages since then. During the latest pregnancy she had eight antisyphilitic treatments, details of which are not known. Her Wassermann reaction was said to have been positive, but the results of recent tests are not available. The baby was delivered at home and was not admitted to the clinic of the Stanford University School of Medicine until January 31, at the age of eighteen days. The baby's Wassermann reaction on January 15 (age

of 2 days) was ++++ (B. H.), but the Kahn reaction was negative. The baby showed no sign of disease except palpable inguinal lymph nodes.

The results of quantitative Wassermann tests are shown in table 5.

CASE 6.—J. W. R. was a boy, born on Jan. 23, 1935, at full term. The weight at birth was 3,380 Gm. The Wassermann reaction of the mother was found to be positive in June 1931, during her first pregnancy, and she was treated; the child had a +++ Wassermann reaction at birth but a negative one at 2 years, having had no treatment. Preceding and during the present pregnancy the mother was treated thoroughly from July 1933; her Wassermann reaction in October 1933, January 1934 and June 1934 was negative, but in October 1934

TABLE 5.—*Results of Quantitative Wassermann Tests (Case 5)*

Date	Age	Amount of Serum, Cc.	Reaction	Units of Reagin
January 31	18 days	0.05	+++	
		0.03	–––	2
February 6	23 days	0.05	++±	
		0.03	–––	2
February 13	30 days	0.1	+++	
		0.07	±––	1
February 21	37 days	0.1	–––	0

TABLE 6.—*Results of Quantitative Wassermann Tests (Case 6)*

Date	Age	Amount of Serum, Cc.	Reaction	Units of Reagin
January 28	5 days	0.1	±––	0+
January 29	6 days	0.1	+++	
		0.07	+++	
		0.03	+––	
		0.01	–––	1.4+
February 9	17 days	0.1	++–	
		0.07	±––	
		0.03	–––	1
February 12	20 days	0.1	–––	
		0.07	–––	0
March 18	55 days	0.1	––– (B.H.)	0

it reverted to ++++ (B. H.). The baby at birth and since then has been in good condition, except for some nasal discharge soon after birth, which did not persist. He was breast fed, and growth was normal. There were no signs of disease.

On April 19 (age 12 weeks) the spinal fluid contained 5.5 leukocytes. The Wassermann reaction was negative, and the colloidal gold curve was 1111000000. The result of a Wassermann test of the mother's milk on Feb. 8, 1935, showed that it was entirely anticomplementary.

CASE 7.—D. O. was a Mexican girl, born on Jan. 30, 1935, at full term. The weight at birth was 8 pounds and 10 ounces (3,910 Gm.). The mother had received antisyphilitic treatment during the preceding three pregnancies (first, seven years previously) and during the latest one was treated weekly at a "pre-

natal clinic" for about eight weeks before delivery. Her last Wassermann reaction before delivery was +++ (B. H.), but she did not show any clinical evidence of syphilis. Fifty hours after delivery her Wassermann reaction determined at the Stanford University School of Medicine was ±———. The baby showed no evidence of disease. The reaction to quantitative Wassermann tests are shown in table 7.

On April 19 the spinal fluid contained 4.4 leukocytes; the reaction to the Nonne and Noguchi tests was negative; the Wassermann reaction was negative; the colloidal gold curve was 1111000000.

TABLE 7.—*Results of Quantitative Wassermann Tests (Case 7)*

Date	Age	Amount of Serum, Cc.	Reaction	Units of Reagin
Jan. 30, 1932	Birth		+++ (B.H.)	At least 1
February 26	4 weeks	0.1	———	
		0.07	———	0
April 19	11 weeks	0.1	——— (B.H.)	0

TABLE 8.—*Results of Quantitative Wassermann Tests (Case 8)*

Date	Age	Amount of Serum, Cc.	Reaction	Units of Reagin
Feb. 14, 1935	Birth	0.1	+++ (B.H.)	At least 1
March 4	18 days	0.05	+++	
		0.03	+——	
		0.01	———	2+
March 28	43 days	0.05	+++	
		0.04	++—	
		0.03	———	2.5
April 10	56 days	0.1	++—	
		0.07	±——	
		0.05	———	1
April 30	76 days	0.1	——— (B.H.)	0

CASE 8.—D. H. was a Chinese girl, born on Feb. 14, 1935; the weight at birth was 7 pounds and 4 ounces (3,285 Gm.). The mother had ten weekly antisyphilitic injections in a "prenatal clinic" during pregnancy; there was no record of her Wassermann reactions. The baby was born at another hospital and was first seen by us at the age of 18 days. She was breast fed and showed no sign of disease. Roentgenograms of the arms and legs on April 9, 1935 (age 54 days), showed no sign of syphilis. She gained well. The results of the Wassermann tests are shown in table 8.

On April 10 the Wassermann reaction of the mother's milk (centrifugated and filtered) was ————.

On April 10 (age 56 days) the Wassermann reaction of the spinal fluid was negative; tests of blood and other tests were not made.

TWO CASES OF TRUE CONGENITAL SYPHILIS

CASE 9.—T. was born on May 18, 1935, at full term, weighing 7 pounds (3,170 Gm.). The mother was unmarried; she had had frequent sexual relations for the last five years but was never pregnant before. She showed no obvious lesions of syphilis, and had received no treatment during pregnancy. The reactions to Wassermann tests on the day of birth are shown in the following tabulation:

Amount of Serum, Cc.	Reaction	Units of Reagin
	Mother	
0.01	+ + +	
0.005	+ + −	
0.0025	− − −	20 −
	Infant (Blood from Cord)	
0.000625	+ + +	160 +

All dilutions of serum down to 0.000625 gave a + + + reaction

The infant died on May 20, aged 2 days. Necropsy showed pneumonia alba and an enlarged liver and spleen. The report of microscopic examination was not yet available at the time of writing.

CASE 10.—A. was a Mexican, born prematurely on May 15, 1935, weighing 4 pounds (1,810 Gm.). Nothing was known of the father. The mother had two older, apparently healthy children. She was not known to be syphilitic, having had no antepartum supervision and no treatment. The baby at birth had universal pemphigus and a palpable liver and spleen; it died a few hours later. Necropsy showed pneumonia alba and an enlarged liver and spleen; the full details of the necropsy, including the histologic examination, are not yet available at the time of writing. The results of Wassermann tests on the day of delivery are shown in the tabulation.

Amount of Serum, Cc.	Reaction	Units of Reagin
	Mother	
0.01	+ + +	At least 10
(No higher dilutions were tested)		
	Infant	
0.00125	+ + +	
0.000625	+ + −	160

COMMENT

While the number of cases is small and will, it is hoped, be increased later by ourselves and others, it is clear that the quantitative Wassermann test gives an earlier clue to the absence of syphilitic infection than does the ordinary simple qualitative test. In seven of the eight presumptively nonsyphilitic babies the reaction to the initial routine Wassermann test was + + +, but with the exception of that in case 3, the initial titer was low, giving an immediate hint—though not, of course, a proof—that actual syphilis might not be present. This is in sharp contrast with the high unitage in the two syphilitic babies. The infant in case 3, with its initial high titer, however, was strongly suspected of syphilis and might have justifiably, even without clinical evidence, been subjected to treatment had not the test on the seventh day shown a sharp reduction of the number of units of reagin to

one-half the original figure. In this case (which might have been regarded as an instance of third generation syphilis if quantitative observations of the serum had not been available) the progressive but very gradual decline was striking. When determined by the ordinary method, the Wassermann reaction remained + + + as late as the fifty-second day and was still + + — on the seventy-third. In other words, the clue of eventual negativity was given by the quantitative method on the seventh day and not until the seventy-third by the routine method.

We are aware that complete and final proof that some or all of these infants may not still harbor syphilis in latent form has not been offered. They are serologically and clinically normal but must, of course, continue under periodic observation. We feel fully justified in withholding treatment in these and similar cases, first, because no criterion remains for judging either the necessity of treatment or its results and second, because, as has already been stated, arsphenamine therapy has serious risks, to which a nonsyphilitic person should not be subjected. These risks are, in our opinion, greater than those of failing to treat a possibly but improbably present latent infection.

In conclusion, we seriously propose that the transmission of syphilitic reagin from mother to fetus without the transmission of syphilis itself is a soundly established phenomenon and one of sufficiently fundamental importance to be dignified as a law and that this law be designated by the name of its discoverer, Fildes. In view of the development since his time of other methods than the Wassermann test for the demonstration of syphilitic reagin in the serum, the wording might be altered a little from that of his original statement, for example: "Syphilitic reagin in the blood of the new-born infant is diagnostic not of syphilis in the infant but of syphilis in the mother."

CONCLUSIONS

Additional proof is offered that syphilitic reagin in the blood of the new-born infant is diagnostic not of syphilis in the infant but of syphilis in the mother. For this phenomenon we propose the name "Fildes' law," from its discoverer.

In the absence of definite evidence of syphilitic infection, a positive reaction to the ordinary qualitative test is not sufficient justification for subjecting the infant to the risks of arsphenamine therapy.

For infants with an initially positive reaction to tests made by the routine method, the quantitative method, in which the amount of reagin present is measured, is a valuable means of distinguishing between those who have and those who have not syphilitic infection. In the

latter, the amount of reagin is usually, but not always, small and, in any case, progressively declines to zero.

The decline occurs early and, in our experience, can be detected by the end of the first week or sooner, whereas a Wassermann test made by the ordinary method may fail to show any evidence of weakening till much later. In one case, the titer was found to be declining on the seventh day but did not reach zero until the ninety-fourth day, and the reaction to the routine test remained "strongly" positive (+++) until the seventy-third day.

METABOLISM OF ADOLESCENT GIRLS

III. THE EXCRETION OF CREATININE AND CREATINE

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CINCINNATI

Sixty-six years have elapsed since Hofmann [1] in 1869 found that the excretion of creatinine varies with age, weight and sex. Although there have been numerous investigations based on the problem of creatinine and creatine, there are few data on the excretion of these in adolescence, the period during which the metabolism of creatinine and creatine may undergo the greatest change. Rose [2] reported values for the creatinine and creatine excreted by adolescent girls, but his study was not made on twenty-four hour samples of urine. Light and Warren [3] made an excellent study of the excretion of creatinine and creatine in adolescent boys. We have been able to find only few scattered data on that of adolescent girls between the ages of 12 and 15 years. In view of this fact, we have studied, in the course of our other metabolic investigations, the excretion of creatine and creatinine in adolescent girls.

METHOD OF EXPERIMENTS

Twenty-three girls, aged 12 to 15 years, the same girls who served as subjects for an investigation of the metabolism of fat and protein, were studied. The technic of feeding and the collection of specimens have been described.[4] Folin's [5] method was employed for the determination of creatinine and creatine. Trinitrophenol was purified by Benedict's method.[6] Duplicate determinations were made

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6. Benedict, S. R.: A Method for the Purification of Picric Acid for Creatinine Determinations, J. Biol. Chem. **54**:239 (Oct.) 1922.

on twenty-four hour specimens for the six consecutive days when the girls were on carefully weighed diets. In order to save space only the range and the average of the values of the six day period are given in the tables.

OBSERVATIONS

Creatinine Coefficient.—As shown in table 1, the averages of the creatinine excretion per twenty-four hours increase in a general way with the body weight, ranging from 638 to 1,216 mg., with an average for the group of 883 mg. The creatinine coefficient, i. e., the milligrams of creatinine per kilogram of body weight, runs from 19.5 to 26.4 mg., averaging 22.8 mg. When our results are compared with those found for children of both sexes, our values are higher than the creatinine coefficient, 15.4, reported by Krause [7] and those reported by Harding and Gaebler [8] and by Wang and her associates,[9] but in the majority of cases the earlier investigations included children of much younger age. The oldest subject reported by Harding and Gaebler was only 9½ years of age, and in the previous series of Wang [9] and her associates most of the children were younger than 12 years. Since it has been established that the creatinine excreted increases with increasing age and weight, being an index of the muscular development of the individual subject, higher values would be expected for our subjects. When our values are compared with those for normal women, they are decidedly higher than those reported by Tracy and Clark,[10] who gave an average creatinine coefficient of 15.7 mg. for a group of twenty-six women medical students. Our average is somewhat higher than the average, 18.1, noted by Palmer, Means and Gamble [11] in their experiments on nine nurses between 20 and 23 years of age. However, our values are closely comparable to those of Dye [12] and McLaughlin and Blunt,[13] whose average

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9. (*a*) Wang, C. C.; Frank, M.; Kern, R., and Hays, B. B.: Metabolism of Undernourished Children: III. Urinary Nitrogen with Special Reference to Creatinine, Am. J. Dis. Child. **32**:360 (Sept.) 1926. (*b*) Wang, C. C., and Kaucher, M.: Metabolism of Undernourished Children: VIII. The Effect of High and Low Protein Diets on the Excretion of Creatine, Creatinine and Ammonia, ibid. **38**:468 (Sept.) 1929.

10. Tracy, M., and Clark, E. E.: Excretion of Creatinine by Normal Women, J. Biol. Chem. **19**:115, 1914.

11. Palmer, W. W.; Means, J. H., and Gamble, J. L.: Basal Metabolism and Creatine Elimination, J. Biol. Chem. **19**:239, 1914.

12. Dye, M.: Basal Metabolism of Normal Women and Its Relation to Creatine and Creatinine Elimination, Thesis, University of Chicago, 1922.

13. McLaughlin, L., and Blunt, K.: Some Observations on the Creatinine Excretion of Women, J. Biol. Chem. **58**:285 (Nov.) 1923.

TABLE 1.—*Excretion of Creatine and Creatinine per Twenty-Four Hours, Average for Six-Day Periods*

Subject*	Age		Weight, Kg.	Preformed Creatinine				Ratio of Creatinine Nitrogen to Basal Metabolism, Mg. per Cal.	Nitrogen Intake per Mg. of Creatinine Excreted, Mg.	Nitrogen Retention per Mg. of Creatinine Excreted, Mg.	Creatine				Total Creatinine	
				Average Total, Mg.	Per Kg.						Average Total, Mg.	Per Kg.			Per Kg., Mg.	Ratio of Total Creatinine to Basal Metabolism, Mg. per Cal.
	Yr.	Mo.			Minimum, Mg.	Maximum, Mg.	Average, Mg.					Minimum, Mg.	Maximum, Mg.	Average, Mg.		
17	13	7	27.94	664	23.5	24.1	23.8	0.254	22.1	2.6	530	18.1	19.8	19.0	42.8	1.22
8	12	3	29.06	638	16.4	24.1	21.8	0.226	17.4	4.9	148	3.4	7.7	5.1	26.9	0.74
6	12	1	30.46	766	24.3	26.2	25.1	0.264	18.9	1.2	432	12.5	16.2	14.2	39.3	1.11
11	12	8	30.72	745	24.0	24.6	24.2	0.241	17.0	1.5	541	16.1	18.5	17.6	41.9	1.11
13	12	9	32.85	777	23.0	24.1	23.7	0.264	15.6	1.5	351	9.1	12.0	10.7	34.3	1.03
3	11	6	32.95	748	22.0	23.6	22.7	0.230	19.9	2.1	488	13.8	15.9	14.8	37.5	1.02
35	13	4	34.45	910	22.9	29.4	26.4	.....	21.1	5.3	502	11.1	18.6	14.9	41.3	....
12	12	8	35.64	771	21.0	22.9	20.4	0.230	19.3	2.1	503	12.3	15.6	14.1	34.5	0.99
5	12	1	36.25	847	22.2	24.4	23.4	0.275	18.0	2.1	490	13.0	13.7	13.5	36.9	1.17
7	12	3	36.27	779	15.8	25.4	21.5	0.227	19.9	3.4	392	5.2	14.0	10.8	32.3	0.92
15	13	2	37.19	826	20.1	23.8	22.2	0.232	14.7	4.1	391	9.1	13.1	10.5	32.7	0.93
20	13	8	37.40	764	17.5	22.5	20.4	0.237	20.1	4.1	424	7.9	15.1	11.3	31.8	0.97
14	12	9	38.79	913	22.8	24.3	23.5	0.263	16.6	0.8	520	12.3	14.0	13.4	36.9	1.12
22	14	6	40.25	1,003	24.2	26.0	24.9	0.292	16.7	3.3	388	8.9	10.1	9.6	34.6	1.10
10	12	5	40.66	853	20.5	21.3	21.0	0.261	18.2	1.1	600	13.3	16.7	14.8	35.7	1.20
4	11	10	41.18	1,000	23.6	24.7	24.3	0.298	15.6	1.3	372	8.2	9.8	9.0	33.3	1.11
9	12	3	41.78	941	22.1	22.8	22.5	0.276	15.8	1.7	395	6.4	12.9	9.5	32.0	1.06
23	14	6	43.83	1,063	23.5	25.0	24.3	0.332	14.5	1.6	247	5.0	6.5	5.6	30.2	1.12
16	13	5	43.94	898	12 2	23.8	20.4	0.244	17.1	2.3	399	4.6	11.1	9.1	29.5	0.95
18	14	5	46.80	1,030	21.6	22.4	22.0	0.267	14.9	0.7	541	9.7	12.6	11.6	33.6	1.10
19	13	2	47.48	1,114	21.1	25.4	23.5	0.317	14.8	3.4	289	4.2	9.0	6.1	29.6	1.04
24	14	7	50.91	1,216	21.0	25.3	23.9	0.308	12.2	0.4	429	7.7	9.5	8.4	32.3	1.12
26	15	3	52.89	1,034	15.1	21.8	19.5	0.277	14.6	2.1	186	1.3	5.7	3.5	23.0	0.88
Average	13	1	38.68	883			22.8	0.264	17.2	2.3	416			11.2	34.0	1.05

* The subjects are arranged according to increasing body weight.

creatinine coefficients are 21.6 and 20.3 mg., respectively. When our results are compared with those for adolescent boys and men, our average is in close agreement with the value 23.09 given by Garot [14] for his subjects between 15 and 30 years of age, and it lies in the range between 21.6 and 29.7 mg. which is obtained by recalculating the values given by Shaffer [15] for men. Our average is slightly lower than those for adolescent boys on uncontrolled diets reported by Light and Warren. Their averages for creatinine are 25.2, 24.9, 25.6 and 25.8 mg. for the different age groups between 14 and 18 years, respectively. Several investigators [16] have pointed out that the somewhat lower coefficient for women is due to their lower degree of muscular development as compared with that of men, and that athletic women have values as high as those of men.

The high values reported for adolescent subjects in our study as well as in that of Light and Warren suggest the possibility that during adolescence girls and boys may have creatinine coefficients as high as those of adults, or higher. This idea receives support from Garot's [14] studies in which he found that the coefficient increased progressively in his age groups (½ to 2 years, 5 to 10 years, 15 to 30 years, 40 to 50 years and over 50 years) to a maximum of 23.09 mg. for the 15 to 30 year old group and then declined rapidly to 16.30 mg. for the group over 50 years of age, a value comparable to that of infancy.

Relation Between Nitrogen Ingested and Creatinine Excreted.—The nitrogen intake per milligram of creatinine excreted in our subjects varies widely, ranging from 12.2 to 22.1, with an average of 17.2 mg. These values are decidedly lower than the figures reported by Daniels and her associates,[17] which ranged from 20 to 49 mg. However, hers were for children from 3 to 6 years old. The one set of figures available in her paper gave from 30 to 34 mg. of nitrogen intake per milligram of creatinine excreted for children receiving 3.2 Gm. of protein per kilogram. Our girls received an average of 2.5 Gm. of protein per kilogram. In view of this wide difference in the ratio of nitrogen ingested to creatinine excreted for different age groups it seems evident that before creatinine excretion can be used as an index for estimating

14. Garot, L.: L'excrétion de la créatinine et la grandeur de la dépense énergétique dans l'espèce humaine, Arch. internat. de physiol. **29**:326, 1927.

15. Shaffer, P.: Excretion of Kreatinin and Kreatin in Health and Disease, Am. J. Physiol. **23**:1 (Oct.) 1908.

16. (*a*) Hodgson, P., and Lewis, H. B.: Physical Development and the Excretion of Creatine and Creatinine by Women, Am. J. Physiol. **87**:288 (Dec.) 1928. (*b*) Shaffer.[15] (*c*) Tracy and Clark.[10]

17. Daniels, A. L.; Hutton, M. K.; Knott, E. M.; Wright, O. E.; Everson, G. J., and Scoular, F.: A Study of the Protein Needs of Preschool Children, J. Nutrition **9**:91 (Jan.) 1935.

protein requirement it will be necessary to obtain adequate data concerning the relationship between protein intake and creatinine output at all age and weight levels.

Relation Between Nitrogen Retention and Creatinine Excretion.—The nitrogen retention per milligram of creatinine in the urine in our subjects shows an even wider variation than the nitrogen intake, ranging from 0.4 to 5.4 mg., and demonstrates little relation to the nitrogen intake, although, when expressed as milligrams per kilogram of body weight the nitrogen retention varies with the nitrogen intake, as has been pointed out in a previous communication.[18] When creatinine is used as the basis this relation becomes much less evident, as is shown by the coefficient of correlation between the two, $+0.424 \pm 0.115$ (table 2). This is contrary to the finding of Daniels, who demonstrated a marked relationship between the two. In her study,

TABLE 2.—*Coefficients of Correlation*

Total nitrogen intake per 24 hours to total preformed creatinine	$+0.476 \pm 0.109$
Nitrogen intake per Kg. per 24 hours to preformed creatinine per Kg.	$+0.473 \pm 0.109$
Total nitrogen intake per 24 hours to total creatine	$+0.263 \pm 0.131$
Nitrogen intake per Kg. per 24 hours to creatine per Kg.	$+0.706 \pm 0.071$
Nitrogen intake per mg. creatinine to nitrogen retention per mg. creatinine	$+0.424 \pm 0.115$
Body weight to the ratio $\frac{\text{creatinine nitrogen}}{\text{basal metabolism}}$	$+0.657 \pm 0.082$
Total creatinine per Kg. to basal metabolism per Kg.	$+0.568 \pm 0.097$

as the nitrogen intake per milligram of creatinine decreased, the nitrogen retention per milligram of creatinine likewise went down. This apparent discrepancy between the findings of the two studies emphasizes again the need for more data concerning age factors. The growth needs are entirely different between children 3 to 6 years of age and girls approaching adult stature.

Relation Between Creatinine and Basal Metabolism.—In spite of the high creatinine coefficient observed in our subjects, the average ratio, 0.264 mg. of creatinine nitrogen per calory of basal metabolism, is closely comparable to the average value, 0.294, obtained by recalculating the findings on nine women by Palmer and his co-workers. However, our values show a much wider range, from 0.226 to 0.332 mg., than theirs, from 0.270 to 0.315 mg. This may be due, in part, to the greater variation in the body weight of our subjects. Palmer and others found that the average value in milligrams of creatinine nitrogen per calory of basal metabolism of their eight men was 0.374, which is decidedly

18. Wang, C. C.; Hogden, C., and Wing, M.: Metabolism of Adolescent Girls: II. Fat and Protein Metabolism, Am. J. Dis. Child. **51**:1083 (May) 1936.

higher than that of their women. Smuts,[19] working on animals, also showed that this ratio varies with the size of the animals. Thus, his values are 0.095 for mice, 0.141 for rats, 0.131 for guinea-pigs, 0.304 for rabbits and 0.378 for pigs. The fact that the ratio of creatinine nitrogen in milligrams to basal metabolism in calories increases with increasing weight may be explained by the conclusion of Brody, Procter and Ashworth [20] that creatinine nitrogen does not vary as much with basal metabolism as it does with body weight. That there is some relation of body weight to the ratio of creatinine nitrogen to basal metabolism is evident in our study by the coefficient of correlation $+0.657 \pm 0.082$. Our ratio is slightly lower than the value 0.326 predicted by Brody and his collaborators for animals weighing 40 Kg. (approximately the average weight of our girls). This discrepancy is probably due to the presence of larger amounts of adipose tissue in the girls than in the animals. Palmer and his associates have attributed the difference in the ratios between men and women to the presence of fat in the latter.

Creatine Excretion.—Creatinuria was found in all of our girls. The values for creatine excretion vary greatly, with a minimum of 148, a maximum of 600 mg. and an average of 416 mg. per twenty-four hours. The total creatine per twenty-four hours bears little relation to age, weight or nitrogen intake (table 2). Nor does it show a marked relation to nitrogen retention, although Garot [21] found in his controlled experiments on pigs that the creatine excretion decreased as the nitrogen retention increased while creatinine remained stationary, thereby indicating that the metabolism of exogenous protein gives rise to creatine.

The creatine excretion per kilogram of body weight decreases from 19 to 3.5 mg. as the body weight increases from 27.6 to 53 Kg. Age, per se, seems to have less influence than weight, although the average for the 15 year old group, 6.3 mg., is lower than the averages for the 12, 13 and 14 year old groups, which are 11.5, 11.7 and 14 mg., respectively. Possibly our age range is too small for age in itself to show the marked influence on creatine excretion noted by Rose,[2] Harding

19. Smuts, D. B.: The Relation Between the Basal Metabolism and the Endogenous Nitrogen Metabolism, with Particular Reference to the Estimation of the Maintenance Requirement of Protein, J. Nutrition **9**:403 (April) 1935.

20. Brody, S.; Procter, R. C., and Ashworth, U. S.: Growth and Development with Special Reference to Domestic Animals: XXXIV. Basal Metabolism, Endogenous Nitrogen, Creatinine and Neutral Sulphur Excretions as Functions of Body Weight, Research Bulletin 220, Agric. Exper. Stat., University of Missouri, College of Agriculture, 1934.

21. Garot, L.: L'élimination des corps créatiniques en fonction de la rétention azotée au cours de la croissance, Arch. internat. de physiol. **29**:65, 1927.

and Gaebler [22] and Garot [14] and by Wang, Kern and Hays [9] in their work on undernourished children. Although, as stated previously, little relation is found between the total twenty-four hour creatine and the nitrogen intake, the twenty-four hour creatine per kilogram increases markedly as the nitrogen intake per kilogram increases. The coefficient of correlation is $+0.706 \pm 0.071$. The influence of nitrogen intake on creatine excretion has been definitely shown by a number of investigators [23] and was also demonstrated by Wang and Kaucher [9b] in a previous report on the effect of high and low protein diets on children. Thus, in a group of eight normal subjects, when the average protein intake was equivalent to 0.56 Gm. of nitrogen per kilogram of body weight the average creatine excretion was 16.3 mg. per kilogram; and when the protein intake was lowered to 0.27 Gm. of nitrogen per kilogram the creatine excretion was almost proportionately lowered to 7.2 mg. This effect was undoubtedly due to the presence of one or more precursors of creatine in the proteins used.

Total Creatinine (Preformed Creatinine Plus Creatine).—The total creatinine (preformed creatinine plus creatine) excreted per kilogram per twenty-four hours varies from 23 mg. in a 15 year old moderately obese girl to 42.8 mg. in a 12 year old undernourished girl. The average value for the group is 34 mg. In his study of twenty-four male subjects between 7 months and 70 years of age, Garot [14] found that the total creatinine excretion remains practically constant up to adult age, when it begins to decline. On a meat-free diet the averages of the total creatinine excreted per kilogram of body weight in his subjects were 27.5 and 27.2 mg. for the two groups from 7 months to 2 years and from 5 to 10 years of age, respectively. The value for the group between 15 and 30 years of age was 24.8 mg., and the values for the group from 40 to 50 and the one above 50 years were 21.4 and 16.8 mg., respectively. Harding and Gaebler,[8] studying six boys and six girls from 2½ to 9½ years of age who were on a meat-free diet, and

22. (*a*) Harding, V. J., and Gaebler, O. H.: The Influence of the Positive Nitrogen Balance upon Creatinuria During Growth, J. Biol. Chem. **57**:25 (Aug.) 1923. (*b*) Harding and Gaebler.[8]

23. (*a*) Talbot, F. B., and Gamble, J. L.: The Protein Metabolism of an Infant, Am. J. Dis. Child. **12**:333 (Oct.) 1916. (*b*) Denis, W., and Kramer, J. G.: The Influence of Protein Intake on Creatine Excretion in Children, J. Biol. Chem. **30**:189, 1917. (*c*) Gamble, J. L., and Goldschmidt, S.: A Study of Creatinuria in Infants: The Relation of Protein Intake to Urinary Creatine, ibid. **40**:215, 1919. (*d*) Harding, V. J., and Young, E. G.: Exogenous and Endogenous Nature of the Creatinuria in the Growing Dog, ibid. **41**:XXXV, 1920. (*e*) Garot, L.: L'excrétion des corps créatiniques au cours de l'inanition protéique, Arch. internat. de physiol. **29**:55, 1927. (*f*) Terroine, E. F., and Danmanville, P.: La formation de la créatine aux dépens des substances protéiques, Compt. rend. Acad. d. sc. **188**:1439 (May 27) 1922.

Light and Warren, studying adolescent boys between 14 and 19 years of age who were on an uncontrolled diet, also pointed out the constancy of total creatinine excretion in their subjects. The average values given by the former authors for the different age groups vary from 22.1 to 23 mg. per kilogram per twenty-four hours, while those given by the latter authors run from 26.4 to 27.9 mg. Thus, not only is our average total creatinine markedly higher than those reported by the aforementioned authors, but our individual variations are much greater than theirs. The only possible explanation we are able to offer for this discrepancy is that the metabolism of creatine in an adolescent girl is different from that in an adult, which is supported by the statement of Krause: "In other words, the power to assimilate creatine is very much weaker in children than in adult men." Our girls were on a self-selected diet with a large proportion of protein derived chiefly from meat, milk and eggs. Since, in the experiments of Rose and his collaborators,[24] female subjects excreted more unchanged creatine than male subjects following oral administration of the compound, it is probable that our girls failed to metabolize the ingested creatine as effectively as would boys and possibly some adult women.

Comparing total creatinine excretion per kilogram with basal metabolism per kilogram in the girls, we find a coefficient of correlation of $+ 0.568 \pm 0.097$. The ratio between the total creatinine per kilogram in milligrams and the basal metabolism per kilogram in calories varies from 0.74 to 1.22, with an average of 1.05. Garot [14] demonstrated a close relationship between the two in his male subjects during the years following childhood and throughout adult life until after the age of 50. The ratio between total creatinine excretion in milligrams and basal metabolism in calories in his 15 to 30 year old group is 0.95, which is not far from our average. According to his theory the creatinine comes exclusively from the muscles and not from the organs. Consequently, owing to the rapid development of the muscular system during growth, the rate of the decrease of total creatinine excretion per kilogram is less than that of the decrease in basal metabolism per kilogram. In old age, the muscular system atrophies before the visceral system and therefore total creatinine excretion decreases more rapidly than the total basal metabolism. This comparison, he stated, confirms the idea that the amount of creatinine represents the height of the energy expenditure of the muscles to the exclusion of that of the organs.

24. Rose, W. C.; Ellis, R. H., and Helming, O. C.: The Transformation of Creatine into Creatinine by the Male and Female Human Organism, J. Biol. Chem. **77**:171 (April) 1928.

SUMMARY

The creatinine coefficient of twenty-three adolescent girls between the ages of 12 and 15 years was as high as, or even higher than, that of an adult woman. The average for the twenty-three girls was 22.8 mg.

The nitrogen intake per milligram of creatinine excreted averaged 17.2 mg., which is much lower than the values reported for preschool children. This value varies apparently with the age and weight of the individual subject. The ratio of nitrogen retention to creatinine excretion varied greatly in different subjects and showed little relation to nitrogen intake.

The ratio between creatinine excretion in milligrams and basal metabolism in calories in the girls averaged 0.264.

Creatine was excreted by all of the twenty-three girls, varying from 3.5 to 19 mg. per kilogram of body weight.

The average value of the total creatinine(creatine plus preformed creatinine) excreted per kilogram found, 34 mg., is much higher than any figure reported in the literature, and there is a wide variation in the individual values.

PNEUMONIA IN INFANTS AND IN CHILDREN

A BACTERIOLOGIC STUDY WITH SPECIAL REFERENCE TO CLINICAL SIGNIFICANCE

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After Dochez and Gillespie's [1] differentiation of pneumococci in 1913, Wollstein and Benson [2] reported the bacteriologic findings in 50 infants and children with pneumonia. They isolated group IV pneumococci from 60 per cent and type I pneumococci from only 8 per cent of their patients. In a high percentage of cases of pneumonia in infants and children the condition was shown to be etiologically related to group IV pneumococci by many studies made prior to 1929, when group IV was further differentiated by Cooper [3] and her co-workers. It is now possible to evaluate the various pneumococci from types IV to XXXII, formerly included as members of group IV. Observations concerning the epidemiology and clinical manifestations of the various pneumococci are shown in the charts and tables. We believe that the accurate classification of pneumococci is of more than academic interest.

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This investigation was made possible by a grant from the Commonwealth Fund to the New York University College of Medicine for research in pneumonia during 1931 to 1933. Assistance was also given during the first year by the Altman Foundation and during the last year by the Metropolitan Life Insurance Company.

This study was made under the direction and advice of Dr. William H. Park, director of the Bureau of Laboratories, Department of Health, New York City, and Dr. Charles Hendee Smith, director of the Children's Medical Service, Bellevue Hospital and professor of pediatrics, New York University College of Medicine.

1. Dochez, H. R., and Gillespie, L. J.: A Biologic Classification of Pneumococci by Means of Immunity Reactions, J. A. M. A. **41**:727 (Sept. 6) 1913.

2. Wollstein, M., and Benson, A. W.: Types of Pneumococcus Found in the Pneumonias of Infants and Young Children, Am. J. Dis. Child. **12**:254 (Sept.) 1916.

3. Cooper, G.; Edwards, M., and Rosenstein, C.: Separation of Types Among Pneumococci Hitherto Called Group IV and Development of Therapeutic Antiserums for These Types, J. Exper. Med. **49**:461 (March) 1929.

The bacteriologic study of all patients with pneumonia began in the Children's Medical Service in Bellevue Hospital in 1928, with the work of Plummer, Raia and Shultz,[4] which continued to June 1930. The present report covers approximately five pneumonia seasons, from Jan. 1, 1931, to June 1, 1935.

METHOD

Laryngeal swabs were taken from all patients in whom pneumonia was suspected. The posterior pharyngeal wall was irritated by a long cotton swab, and the mucus coughed up from the bronchus was caught on the swab. Repeated swabs were taken from patients reported to have no pneumococci and from those having pneumococci of type III, VI, or XIX or any of the other types commonly found in normal children.

Each laryngeal specimen was studied by the inoculation of mice, the Sabin[5] slide agglutination technic and streaked blood agar plates being used. The latter technic is necessary in the cases of pneumonia in children because of the importance of streptococci and staphylococci, particularly in infants. During the years 1933-1934 and 1934-1935 Sabin's modification of the Neufeld reaction was used on many specimens. A report evaluating this newer technic in children is to be published.[7]

The bacteriologic results so obtained were correlated, with the results of a study of other body fluids—pus from the ear, blood, pleural fluid and fluid obtained from postmortem punctures of the lung. These punctures were made within a few minutes after death.

The criteria for clinical diagnosis were the same as those previously outlined.[7a] Some of the laryngeal swabs contained more than one type of pneumococcus; other swabs had a pneumococcus with a streptococcus or staphylococcus, the latter in predominance. Such cases were tabulated under the organism which recurred in cultures from subsequent swabs or which was also cultured from other body fluids, such as pleural fluid or blood, or occasionally from pus discharged from the ear. The accompanying organism was considered nonetiologic.

RESULTS

During the period of study there were 1,033 patients with pneumonia, who were from 2 weeks to 13 years of age. Of these, 796 (or 77 per cent) had pneumonia associated with pneumococci alone; from the remainder there were isolated streptococci, staphylococci and influenza or Friedländer bacilli alone or associated with pneumococci.

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4. (*a*) Plummer, N.; Raia, A., and Shultz, S.: Pneumonia in Children: A Bacteriologic Study, Am. J. Dis. Child. **40**:557 (Sept.) 1930. (*b*) Raia, A.; Plummer, N., and Shultz, S.: New Types of Pneumococci in the Pneumonias of Children, Am. J. Dis. Child. **42**:57 (July) 1931.

5. Sabin, A. B.: The Microscopic Agglutination Test in Pneumonia, J. Infect. Dis. **46**:469 (June) 1930.

6. Sabin, A. B.: Immediate Pneumococcus Typing Directly from Sputum by Neufeld Reaction, J. A. M. A. **100**:1584 (May 20) 1933.

7. Vinograd, J.; Nemir, R. L., and Park, W. H.: The Rapid Typing of Pneumococci by the Neufeld Reaction Directly from Laryngeal Swabs from Infants and Children, Am. J. Dis. Child. to be published.

7a. Nemir, R. L.: The Treatment of Pneumonias in Infants and Children with Antipneumococcus Serum, J. Pediat. **3**:827 (Dec.) 1933.

Table 1 is a composite table of the distribution of organisms by age of the patient and diagnosis during the years studied. No bacteriologic work was done during the summer months. There were many more patients with pneumonia during the pneumonia season 1932-1933, as many as during the preceding and the following season together. This increase in 1932-1933 was due to a greater number of cases of pneumonia due to pneumococci, especially to type XIV. That pneumonia due to a single type of pneumococcus may become epidemic was shown by Schroder and Cooper [8] in 1930 with type V; in 1934 Dooley [9] also reported an epidemic of pneumonia associated with the type I pneumococcus in a boy's school. During the first season (1931) in which this study was conducted, there was an increase of pneumonia associated with type XXII; 12 patients were observed as compared with 9 for the following four seasons. Only 15 patients with pneumonia caused by organisms of type II were seen in the first four seasons, but in the last season alone 13 patients had pneumonia caused by type II.

The relative virulence of the types of pneumococci was estimated by observation of (1) the rate of occurrence in patients who did not have pneumonia, (2) clinical types of pneumonia, (3) incidence of complications, particularly empyema, and (4) mortality rate. These factors will be discussed later in this paper.

The most common types of pneumococci found in infancy and childhood were XIV, I and VI, these three types causing the pneumonia in 42 per cent of all cases in this series. Type XIV was the most common in patients under 2 years; it was still common in the preschool group (2 to 5 years) and became less frequent after 5 years. Conversely, type I was the predominant pneumococcus in older children, being rare in patients under 2 years and not common in those of preschool age. Pneumonia associated with type XIX was found almost entirely in patients under 2 years of age, the majority being under 1 year. Scattered throughout the years of childhood was pneumonia associated with organisms of type VI, occurring with somewhat greater frequency in the earlier years and only occasionally between the ages of 9 and 12 years (tables 1 and 5 and chart 1).

Predominant Pneumococci in Patients Without Pneumonia.—There were 425 patients without pneumonia who were studied clinically and bacteriologically. These children had infections, such as acute tonsillitis, pharyngitis, sinusitis and bronchitis. Patients with pleurisy in whom

8. Schroder, M. C., and Cooper, G.: Epidemic of Colds, Bronchitis and Pneumonia Due to Type V Pneumococci, J. Infect. Dis. **46**:384 (May) 1930.

9. Dooley, P.: An Epidemic of Pneumococcus Type I Infection: Read at the Regional Conference of the American Academy of Pediatrics and the Tri-City Pediatrics Group, Oct. 20, 1934.

TABLE I.—*Distribution of Organisms by Age of the Patients and Diagnosis*

Organism*	Age in Years	Lobar Pneumonia, Year					Bronchopneumonia, Year					Lobular Pneumonia, Year					Pneumonia, Variety Undetermined, Year					Empyema, Year					Total Number of Cases of Pneumonia, Year					Diseases Other than Pneumonia, Year					Pleurisy, Year				
		1931	1932	1933	1934	1935	1931	1932	1933	1934	1935	1931	1932	1933	1934	1935	1931	1932	1933	1934	1935	1931	1932	1933	1934	1935	1931	1932	1933	1934	1935	1931	1932	1933	1934	1935	1931	1932	1933	1934	1935
I	Under 2	7	2	2	3	8	..	1	..	2	0	..	..	..	..	..	..	..	1	..	..	..	..	1	2	0	7	3	3	5	8	..	0	..	2	..	..	..	..	1	..
	2 to 13	18	14	34	19	28	..	0	1	..	..	..	..	..	..	..	..	..	..	..	..	..	3	8	9	1	29	14	35	19	28	..	1	1	3	..	..	..	..	1	..
II	Under 2	0	0	..	..	1	..	0	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	0	0	..	0	1	..	..	..	..	..	..	..	..	..	..
	2 to 13	3	2	8	2	12	..	0	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	3	2	8	2	12	..	..	1	1	1	..	..	..	..	..
III	Under 2	..	3	2	2	1	1	2	..	2	2	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	1	5	2	4	3	..	0	2	8	2	..	..	1	..	..
	2 to 13	1	5	8	3	5	..	0	..	0	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	1	1	5	8	3	5	..	1	2	2	4	..	..	1	..	..
IV	Under 2	1	1	1	0	2	2	..	1	1	..	..	..	..	1	..	..	..	..	..	..	..	1	..	0	..	3	1	2	2	2	..	3	6	3	3	..	..	..	..	..
	2 to 13	2	..	5	4	9	..	..	..	1	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	2	3	0	5	5	9	..	0	2	3	2	..	..	..	..	..
V	Under 2	3	0	1	0	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	3	0	1	0	3	..	..	..	..	..	..	..	..	..	..
	2 to 13	12	3	5	7	9	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	1	..	..	13	3	6	7	9	..	..	4	..	2	..	..	1	1	..
VI	Under 2	4	5	11‡	4	8	5	2	10	4	4	..	1	..	1	..	2	..	7	..	..	..	..	..	..	..	11	8	28	9	12	..	7	..	7	8	..	..	..	2	..
	2 to 13	3	10	12	5	9	..	1	1	1	2	..	1	1	..	..	1	..	..	..	..	..	1	..	..	..	4	12	14	6	11	..	3	10	15	11	..	..	1	2	..
VII	Under 2	1	..	8	0	1	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	10	0	1	..	0	3	1	..	..	..	..	0	..
	2 to 13	..	..	4	9	7	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	9	7	..	1	0	2	..	..	..	1	1	1
VIII	Under 2	..	..	2	..	..	1	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	3	..	0	..	0	2	..	1	..	..	..	..	..
	2 to 13	..	..	2	..	1	..	0	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	0	2	..	1	..	1	2	..	1	..	..	..	..	..
IX	Under 2	1	3	2	1	..	1	1	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	2	4	2	2	0	..	..	2	1	1	..	..	2	..	..
	2 to 13	1	1	3	1	2	..	0	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	3	1	2	..	..	3	0	1	..	..	..	..	..
X	Under 2	1	0	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	2	1	1	..	..	..	..	..
	2 to 13	1	1	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	1	0	..	2	2	0	..	..	..	..	..	..
XI	Under 2	2	1	1	1	2	3	2	..	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	3	1	4	2	..	..	1	1	..	..	..	..	..	..
	2 to 13	3	2	..	1	..	1	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	4	3	..	1	..	..	..	1	0	..	..	..	..	..	..
XII	Under 2	..	..	2	..	1	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	0	2	..	1	..	1	..	1	1	..	..	..	1	..
	2 to 13	1	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	1	..	1	2	..	2	..	..	..	..	..
XIII	Under 2	..	0	..	0	1	1	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	0	1	..	1	1	..	..	..	..	..	..	..
	2 to 13	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	0	..	2	..	..	1	..	..	2	..	..	..	..	..
XIV	Under 2	21	9	32	18	23	4	1	7	5	1	1	..	1	..	..	2	..	..	..	..	..	1	1	3	3	28	10	40	23	24	..	4	5	1	..	..	..	..	4	..
	2 to 13	7	5	21	5	8	..	1	..	0	1	..	..	1	..	..	..	..	1	..	..	..	..	..	..	2	7	6	23	5	9	..	0	2	2	..	0	1	..	0	..
XV	Under 2	..	1	1	2	..	1	1	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	1	4	..	..	1	2	4	1	..	..	..	..	..
	2 to 13	..	1	..	..	..	..	0	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	0	1	..	0	..	..	0	1	..	2	..	..	..	..	..
XVI	Under 2	..	..	3	..	4	..	..	2	..	2	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	5	..	7	..	..	1	2	2	..	..	..	..	1	..
	2 to 13	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	..	..	..	1	..
XVII	Under 2	..	..	..	1	1	1	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	2	1	..	..	4	4	..	..	..	..	..	..
	2 to 13	2	1	..	1	..	1	..	1	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	4	1	2	1	..	..	..	3	2	..	..	..	..	..	..
XVIII	Under 2	2	1	1	0	1	1	3	1	1	1	..	..	..	..	..	1	..	..	..	..	0	4	4	2	1	2	..	2	4	2	3	..	0	4	1	1	..	..	1	..
	2 to 13	1	3	4	4	3	..	0	..	0	..	..	..	..	..	..	..	..	..	..	..	1	1	3	4	4	3	..	0	4	1	1	..	..	..	1	..	..	..	..	..
XIX	Under 2	2	2	8	2	1	7	3	9‡	3	3	..	..	1	1	..	..	1	2	1	..	..	..	..	..	1	9	6	20	7	4	..	3	11	7	4	..	..	..	1	0
	2 to 13	..	..	..	1	0	..	..	1	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..

| | | |

pneumonia did not develop are not included in table 3. Only the most frequent types of pneumococci in the series of patients who had pneumonia and in those who had no pneumonia are tabulated in table 3. Table 1 gives the entire distribution of pneumococci cultured from patients who did not have pneumonia.

In table 3 it can be seen that the type XIX pneumococcus occurred with equal frequency in patients with pneumonia and in those without pneumonia. Types VI and III were found more than half as frequently in the patients without pneumonia as in those with pneumonia. Types VI, XIX and III alone were found in over one third of the entire

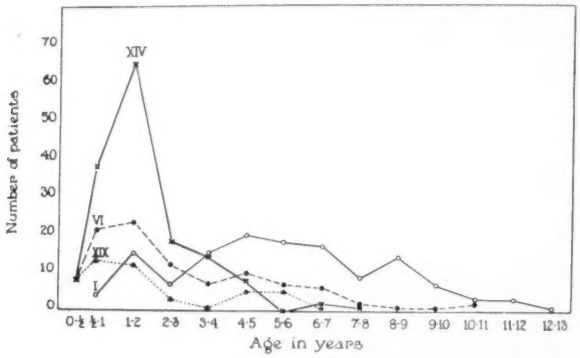

Chart 1.—Age distribution of the predominant types of pneumococci in infants and children. Pneumococci of types XIV and XIX are found predominantly in infants and the run-about group, rarely after 5 years of age. Type I pneumococcus is encountered chiefly in children, being uncommon in infants under 2 years of age. Type VI pneumococcus is most common in infants and children.

series of 425 patients who did not have pneumonia. In contrast was the infrequency with which types I and XIV were found in patients not having pneumonia. In 1932, Cooper and her co-workers [10] found types VI and XIX prevalent in normal persons.

That types III, VI and XIX were often present in the respiratory tracts of sick persons without producing pneumonia was one index of a lower degree of invasiveness of these organisms. The type III pneumococcus in the past had been associated with severe illness and a high mortality rate in adults; type III cross-agglutinated type VIII. An accurate differentiation of types III and VIII has been made for the past two years by using specific rabbit serum in the agglutination tests

10. Cooper, G.; Rosenstein, C.; Walter, A., and Piezer, L.: Further Separation of Types Among Pneumococci Hitherto Included in Group IV and Development of Therapeutic Antisera for These Types, J. Exper. Med. **55**:531 (April) 1932.

and in determining the Neufeld reaction. Type VIII is rare in patients under 13 years of age.

The bacteriologic and epidemiologic observations on healthy carriers in contact with the patients with pneumonia during the year 1931 are in the process of preparation for publication.

Clinical Types of Pneumonia in Relation to Type of Infecting Organisms.—It was noticed early in this study that the clinical picture

TABLE 2.—*Distribution of Dominant Types of Organisms in Patients with Pneumonia*

	Entire Period of Study		Jan. 1, 1931, to Dec. 31, 1931		Jan. 1, 1932, to July 15, 1932		Sept. 15, 1932, to July 15, 1933		Sept. 15, 1933, to May 31, 1934		Sept. 15, 1934, to May 31, 1935	
	Number of Patients	Percentage	Number of Patients	Percentage	Number of Patients	Percentage	Number of Patients	Percentage	Number of Patients	Percentage	Number of Patients	Percentage
Total	1,033		205		142		299		170		217	
Type I	142	13.7	27	13	17	12	38	13	24	14	36	16.5
Type XIV	174	16.8	35	17	16	11	63	21	28	16	32	14.7
Type VI	115	11.1	15	7	20	13	42	14	15	9	23	10.5
Type III	37	3.6	2	1	10	7	10	3	7	3	8	3.6
Type XIX	56	5.4	9	4	6	4	21	7	8	5	12	5.5
All other types	372	36.2	91	45	46	34	99	34	58	35	78	36.3
Streptococci and staphylococci	137	13.2	26	13	27	19	26	8	30	18	28	12.9

TABLE 3.—*Distribution of Prevalent Types of Pneumococci in All Patients Studied*

Type	Patients with Pneumonia		Patients without Pneumonia	
	Number	Percentage	Number	Percentage
I	142	13.7	9	2.1
III	37	3.6	28	6.6
V	45	4.3	6	6.4
VI	115*	11.1	73	17.1
VII	32	3.1	7	1.6
XIV	174	16.8	20	4.7
XVIII	28	2.7	27	6.3
XIX	56	5.4	55	12.9

* Mixed in 5 cases: in 3 with other pneumococci, in 1 with hemolytic streptococci and in 1 with streptococci and staphylococci.

associated with pneumococci differed according to the type of infecting organism. Such a suggestion was made by Cecil and his co-workers [11] in 1927, recently by Sutliff and Finland [12] and repeatedly by Bullowa.[13]

11. Cecil, R. L.; Baldwin, H. S., and Larsen, N. P.: Lobar Pneumonia: Clinical and Bacteriologic Study of Two Thousand Typed Cases, Arch. Int. Med. **40**:253 (Sept.) 1927.

12. Sutliff, W. D., and Finland, M.: Significance of Newly Classified Types in Disease: Type IV to XX Inclusive, J. A. M. A. **101**:1289 (Oct. 21) 1933.

13. Bullowa, J. G. M.: (*a*) Pneumonias Due to Pneumococcus Type VIII, Am. J. M. Sc. **190**:65 (July) 1935. (*b*) Pneumonia Due to Pneumococcus Type XIV (Cooper) and Its Treatment with Specific Antiserum, J. Clin. Investigation **14**:373 (July) 1935.

In our series of patients with lobar pneumonia certain pneumococci, namely, types I, XIV and V, almost always produced a clearcut pneumonia of moderate to high severity, rarely a mild disease. Pneumonia associated with types VI, XIX and III, on the other hand, was usually mild and of short duration and at times was difficult to diagnose without the aid of a roentgenogram. The duration of disease in this last group was several days shorter. The latter types are also the ones found commonly in normal persons and, in our series, in patients sick with disease of the respiratory tract but not with pneumonia. Significant also is the fact that when the types of the first group were associated with pneumonia, they occurred in patients with lobar pneumonia, a disease admittedly related etiologically to pneumococci. Ninety-three per cent of the patients with types I, XIV, VII and V pneumococci had

TABLE 4.—*Clinical Types of Pneumonia According to Age*

	All Ages Under 13 Years			Under 2 Years			2 to 13 Years		
	Number of Patients	Percentage with Pneumococci	Percentage with Other Organisms	Number of Patients	Percentage with Pneumococci	Percentage with Other Organisms	Number of Patients	Percentage with Pneumococci	Percentage with Other Organisms
Lobar pneumonia	758	90	10	286	93.4	6.6	472	88.1	11.6
Bronchopneumonia	209	79.9	20.1	190	79.0	21.0	19	89.5	10.5
Lobular pneumonia	21	....	....	16	....	....	5	....	....
Type undetermined	45	....	....	34	....	....	11	....	....
Total	1,033			526			507		

lobar pneumonia. On the other hand, the less invasive types when associated with pneumonia occurred more commonly in patients with bronchopneumonia, a disease in which the etiologic significance of pneumococci has been frequently questioned. In our series, types III, VI and XIX occurred almost as commonly in patients with nonlobar pneumonia as in those with lobar pneumonia. Although these types have a greater incidence in the younger age group, in which bronchopneumonia is common, this factor is not the only explanation; type XIV is also common in patients under 2 years.

Lobar pneumonia in patients of all ages is primarily of pneumococcic origin; pneumococci were found in 90 per cent of all patients with lobar pneumonia, types I, XIV and VI occurring in 46.7 per cent of the entire series, as shown in table 5.

Bronchopneumonia is a disease mainly seen in infancy, being rare in children of more than 3 years; however, lobar pneumonia in our series occurred even more often in infants than did bronchopneumonia. Lobular pneumonia in our experience is primarily an aspiration pneu-

monia in young or feeble infants, and the bacteriology is not particularly significant; the organisms found in carriers are most often cultured from the swabs from infants with this form of pneumonia.

Bronchopneumonia was associated with streptococci or staphylococci alone in a higher percentage of cases than lobar pneumonia, as shown in table 4. Equally significant was the low incidence of types I, XIV, VII and V in patients with bronchopneumonia, these types occurring in only 13 per cent of the patients as compared with 47.7 per cent of patients with lobar pneumonia. In other words, approximately two thirds of the patients with bronchopneumonia had a disease associated with both streptococci and staphylococci and with the types of pneumococci found in healthy carriers; on the other hand, only one third of the patients with lobar pneumonia fell in this group (mostly type VI).

TABLE 5.—*Predominant Pneumococci in Patients with Lobar Pneumonia*

	All Ages Under 13 Years		Under 2 Years		2 to 13 Years	
Types	Number of Patients	Percentage	Number of Patients	Percentage	Number of Patients	Percentage
I	135	17.8	22	7.7	113	23.9
VI	71	9.3	32*	11.2	39	8.2
XIV	149	19.6	103	36.0	46	9.7
V	44	5.9	7	2.4	37	7.8
VII	32	4.4	12	4.2	20	4.2
XIX	22	2.9	15	5.0	7	1.5
Totals	453	59.9	191	66.5	262	55.3
Other organisms (than pneumococci)	75	10.0	19	6.6	56	11.9
Number of cases	758†	100.0	286	100.0	472	100.0

* Mixed with hemolytic streptococci or with other pneumococci.
† Data for 230 patients not charted; these had lobar pneumonia associated with other types of pneumococci than those listed.

It seems most unlikely that these organisms should have a primary causal rôle in a disease of such severity and fatality as bronchopneumonia. On the other hand, pathologic evidence in some of our cases suggests the etiologic rôle of streptococci and staphylococci as secondary invaders in bronchopneumonia. Pulmonary abscesses and cultures of fluid obtained by puncture of the lung immediately after death, as well as occasional positive results of blood cultures, support the theory that these organisms play an important rôle in some cases. Whether they are sole or only partial factors in the etiology cannot be stated at present. A summary of the bacteriology of bronchopneumonia is given in table 6.

Mixed Types.—In 116 patients with pneumonia and 21 patients who were sick but did not have pneumonia, more than one organism, that is, two or more types of pneumococci or pneumococci and streptococci, were cultured. The possible interpretations, as Finland[14] has pointed

14. Finland, M.: Significance of Mixed Infections in Pneumococcus Pneumonia, J. A. M. A. **103**:1681 (Dec. 1) 1934.

out, are threefold: (1) one organism responsible for disease and the other a normal inhabitant of the throat; (2) the two organisms producing disease simultaneously, and (3) one organism following the other in the production of disease. The majority of our patients were in the first group, with pneumococci of types VI, III and XIX occurring as nonetiologic agents in a high proportion. These three organisms appeared in 54.4 per cent of the patients with mixed infection. Many of the higher-numbered types of pneumococci have not in our experience produced pneumonia and were found commonly in patients without pneumonia; hence when these organisms do occur with the types known to be highly pathogenic, the latter alone are considered responsible for the disease.

Evaluation of the etiologic agent in mixed infections was particularly important in the administration of antipneumococcus serum. The specific therapeutic agent failed to show a beneficial effect at times because of error in etiologic or bacteriologic diagnosis as illustrated by one of

TABLE 6.—*Bacteriologic Study of Bronchopneumonia*

	All Organisms	Type I	Type VI	Type XIV	Type XIX	Other Pneumococci	Hemolytic Streptococci	Streptococcus viridans	Staphylococci
Number of patients....	209	4	30*	20	28*	82†	21	9	15
Percentage.....	100	1.9	14.4	9.6	13.4	39.2	10.0	4.3	7.2

* See table 7—many mixed with streptococci.
† One patient with Friedländer's bacillus type A and type XI pneumococcus.

us [7a] in a study of serum therapy. Because such a failure was often associated with types VI and XIX, pneumococci commonly found in patients without pneumonia, repeated cultures were taken from such patients. Agglutinin tests were not done on the patient's blood.

CASE 1.—D. M., aged 10 years, was admitted to the hospital acutely ill with pain in the chest, fever and vomiting of one day's duration. The type XXX pneumococcus was isolated. Because of the low pathogenicity of this type of pneumococcus, a second swab was taken and showed type VII alone. Antipneumococcus serum of type VII, 19 cc. (1,900 units), was given, and crisis followed eight hours later, on the fourth day of disease.

This case illustrates the importance (1) of matching the pathogenicity of the organism with the severity of the disease, (2) of making repeated cultures in the cases of patients having pneumococci of relatively low pathogenicity and (3) of evaluating the type of pneumococcus in using specific therapeutic agents.

CASE 2.—A. M., aged 1½ years, was admitted with a history of fever, anorexia and restlessness for three days. The temperature was 105 F. for one day. There were signs of lobar pneumonia in the right lower lobe. The type VI pneumococcus was isolated from one culture, and specific serum was given intravenously and intramuscularly on the fifth day of the illness (16 cc., or 25,500

units), without any change in the patient's condition. Empyema was diagnosed three days after serum therapy, and the type V pneumococcus was cultured from the pleural fluid.

This case illustrates the failure to obtain the etiologic agent from one swab and the consequent poor result with antipneumococcus serum. The type VI pneumococcus was most likely a normal inhabitant of the patient's upper respiratory tract.

The type XIX pneumococcus presents a problem in the evaluation of virulence. It occurs in patients not having pneumonia, and yet it is also found in severely ill patients with bronchopneumonia, frequently

TABLE 7.—*Mixed Organisms in Patients with and without Pneumonia*

Types	Number of Patients	Number of Times Probably Causative	Number of Times Probably Not Causative*
I	22	22	0
II	3	3	0
III	22	7	15
IV	15	2	13
V	16	16	0
VI	33	7	26
VII	4	4	0
VIII	8	2	6
IX	11	5	6
X	1	1	..
XI	6	4	2
XII	5	3	2
XIII	6	0	6
XIV	29	25	4†
XV	4	..	4
XVI	8	1	7
XVII	6	4	2
XVIII	14	6	8
XIX	19	2	17
XX	4	2	2
XXI	7	0	7
XXII	10	7	3
XXIII	5	0	5
XXV	2	0	2
XXVIII	3	0	3
XXIX	3	1	2
XXX	6	1	5
XXXI	5	1	4
Hemolytic streptococci	4	4	..

* Pneumococci occurring in patients without pneumonia are all charted under noncausative.
† Two instances in patients not having pneumonia, hence under noncausative; and 2 in patients having hemolytic streptococci, 1 dying after meningitis developed, and the other having empyema from which hemolytic streptococci were cultured. Type XIV may have been causative in the beginning and hemolytic streptococci secondary invaders in the last 2 cases.

associated with hemolytic streptococci. Several explanations are offered: The type XIX pneumococcus may be the preliminary infecting organism and may favor the invasion by hemolytic streptococci; both may be responsible for the disease simultaneously, with greater pathogenicity related to streptococci, or the type XIX pneumococcus may have no relation to the pneumonia and may be only an inhabitant of the upper respiratory tract. Study of the tables of mortality helps in interpreting the rôle of this type in the production of the disease of the lungs.

Complications.—In the reports of the incidence of complications in the past various figures have been cited. The type I pneumococcus has always been a chief agent in the production of empyema. In 1922

Lyon [15] found a 38 per cent incidence in the cases of pneumonia caused by type I pneumococci. Of this series of 1,033 patients, 105 had empyema. Certain organisms, although producing pneumonia, did not in our experience produce empyema.

In table 8 types I and V stand out as the predominant pneumococci producing empyema, which occurred in 21.1 per cent of the patients with pneumonia caused by type I and in 24.4 per cent of the patients with pneumonia caused by type V. These two types represent 38 per cent of all the patients with empyema. The hemolytic streptococcus also

TABLE 8.—*Bacteriology of Complications*

Organism	Number of Patients with Pneumonia	Empyema		Positive Blood Cultures		Meningitis	
		Number of Patients	Number of Deaths	Number of Patients	Number of Deaths	Number of Patients	Number of Deaths
Total	1,033	105	25	16	13	17	17
I	142	30	2	1	..	..	..
II	28	2	0	..	..	..	..
III	37	4	..	..	..	..	..
IV	31	2	..	..	..	..	..
V	45	11	3	2	2	1	1
VI	115	2	1	4	4	4	4
VII	32	2	1	1	..	..	..
IX	18	1	..	1	1	1	1
XI	23	2	1	1	1	1	1
XII	8	0	..	2	1	1	1
XIV	174	14	5	..	..	1	1
XVI	12	0	0	..	..	1*	1*
XVII	11	1	1	..	..	..	..
XVIII	28	1	0	..	..	1	1
XIX	56	1	..	..	..	..	..
XXI	12	1	..	..	..	1	1
XXIII	14	1*	..	..	..	..	..
Hemolytic streptococci	75	14	5	2	2	2†	2
Streptococcus viridans	40	4	1	..	..	..	..
Staphylococcus	26	6	4	2	2	..	..
Pneumococcus, unclassified	8	1	..	..	..	2‡	2
Sterile	..	4	1	..	..	..	..
Influenza	1	1	..	..	..	1	1

* Combined with hemolytic streptococci.

† Swab mixed with pneumococci; 1 with type XVI; 1 with type XVIII. Spinal fluid showed only hemolytic streptococci.

‡ For the spinal fluid from one patient, diagnostic serums were available for types I to XX only.

has a high incidence (19 per cent) and is associated with a high death rate in the series of cases of empyema. In our study empyema occurred in a somewhat higher ratio in 1933-1934 than in the previous three years.

It can be seen that although empyema developed in 10 per cent of all patients with pneumonia, this statement is far from giving the correct impression. Many types of pneumococci have not been associated with empyema, while other types are followed by empyema in a much higher ratio than 10 per cent, the general figure for the entire series. Small pulmonary abscesses developed in two patients with pneumonia

15. Lyon, A. B.: Bacteriologic Study of Pneumonia and Post-Pneumonic Empyema, Am. J. Dis. Child. **23**:72 (Jan.) 1922.

associated with pneumococci, in one with type VII and in the other with type I. Both these patients recovered.

Blood cultures were taken only from patients who were severely ill. Positive results were obtained for 15 of the 98 patients studied. A higher ratio was obtained in this picked group of severely ill patients than would be found if cultures were made from the blood of all

TABLE 9.—*Analysis of Deaths Associated with Pneumococci Types XIX and VI* *

	Number of Patients				
Type in Throat Swab	Total	Specific Pneumococci Alone in PMLP	Specific Pneumococci with Streptococci and Staphylococci in PMLP	Only Streptococci or Staphylococci in PMLP	No PMLP Done
XIX	16	1	2	6	7
VI	21	5	5	6	5

* PMLP means fluid obtained by postmortem lung puncture.

TABLE 10.—*A Comparison of Distribution of Mortality During the Pneumonia Seasons Studied*

			Type of Infecting Organism*				Percentage of Mortality		
			Pneumococci, Types I-XXXII		Streptococci and Staphylococci				
Dates of Pneumonia Seasons	Total Number of Cases	Total Number of Deaths	No. of Patients	Deaths	No. of Patients	Deaths	Total	Pneumococci, Types I-XXXII	Streptococci and Staphylococci
Jan. 1, 1931, to Dec. 31, 1932	205	47	179	35	26	12	22.9	19.6	46.1
Jan. 1, 1932, to July 15, 1932	142	43	113	27	29	12	30.2	23.9	41.4
Sept. 15, 1932, to July 15, 1933	299	49	273	24	26	13	16.4	9	50
Sept. 15, 1933, to May 31, 1934	170	24	142	8	28	16	14.7	6	57
Sept. 15, 1934, to May 31, 1935	217	23	189	11	28	10	10.6	5.8	35.7

* It is difficult to determine the exact causative organisms when mixed infections are present. In this group we considered only those pneumococci which were either pure or were mixed with streptococci and staphylococci, except types VI and XIX, which were studied in table 9. Eighteen of the cases of mixed infection were not included; 4 in 1932, 12 in 1932-1933, and 2 in 1934-1935.

patients. All but 3 of these patients died. The 3 who survived were treated with specific serums, one for type XIV, another for type I and the third for type VII; however, 4 of the other patients received specific serums (all type VI) but did not survive.

Pneumococcic meningitis occurred in 17 patients, all of whom died; 3 of the 4 patients with type VI meningitis were given specific intravenous and intrathecal injections of antipneumococcus serum. All these patients except 2 were under 2 years of age, and all but two had blood cultures which contained pneumococci. Meningitis as an early compli-

cation with septicemia occurred as commonly as it did as a late one. The absence in the patients with meningitis of the type I pneumococcus, an organism of relatively high pathogenicity, may be accounted for on the basis of the age of patients with meningitis; the type I pneumococcus is rarely found in patients under 2 years.

TABLE 11.—*Distribution of Deaths by Infecting Organisms*

Organism	Number of Patients with Pneumonia	Numer of Deaths	Comment*
I	142	9	Two mixed, 1 with type IX pneumococci and 1 with type XI
II	28	1	
III	37	5	Two mixed with hemolytic streptococci
V	45	2	
VI	115	21	See table 9
VII	32	2	PMLP revealed hemolytic streptococci in 1 patient, and autopsy showed pulmonary abscesses
VIII	9	2	
IX	18	4	
XI	23	6	
XII	8	4	
XIII	5	2	One had lipoid pneumonia on autopsy; PMLP showed staphylococci; excluded from mortality figures
XIV	175	22	
XVI	12	3	All mixed with hemolytic streptococci in swab; only hemolytic streptococci in PMLP of one patient, second patient ultimately died of streptococcic meningitis
XVII	11	1	
XVIII	28	7	One mixed with hemolytic streptococci which produced streptococcic meningitis
XIX	56	16	See table 9
XXI	12	2	
XXII	21	1	
XXIII	16	3	
XXIV	5	1	Mixed with hemolytic streptococci
XXVIII	7	1	Mixed with hemolytic streptococci in swab; only hemolytic streptococci in PMLP
XXIX	9	1	
XXX	4	1	
XXXI	10	1	
Unclassified pneumococci	9	3	
Hemolytic streptococcus, staphylococcus, Streptococcus viridans	136	62	
Friedländer A and type XI pneumococcus	1	1	
Influenza bacillus	1	1	
Unknown	..	1	

* PMLP means postmortem lung puncture.

In cases of acute otitis media during pneumonia, when cultures were made from material on the knife immediately after myringotomy, pneumococci were obtained in approximately 70 per cent of the cases. Cultures made later, however, were usually found to yield staphylococci. Purulent otitis media was a common complication in infants and relatively uncommon in older children. It was not surprising, therefore, that the type XIV pneumococcus, a type commonly found in infants, was cultured most frequently from the ears of patients with pneumonia;

type VII was next in frequency. However, otitis media developed in only 10 per cent of the patients with pneumonia due to type XIV, as compared with 25 per cent of those with pneumonia due to type VII. Occasional cases of otitis media associated with pneumonia due to types I, III, XIX, VI and XVIII were observed.

Mortality.—From this study it became apparent that the outcome of pneumonia was dependent on the age of the patient, the type of infecting organism and the type of pneumonic process, i. e., bronchopneumonia, lobar or lobular pneumonia or a combination of these. The mortality rate for the entire series of 1,033 patients was 18 per cent.

TABLE 12.—*Results of Bacteriologic Study in the Children's Medical Service Bellevue Hospital: Predominant Types of Pneumococci*

Author	Year	Number of Patients: All Cases	Type: I	II	III	V	VI	VII	XIV	XIX	Empyema; Percentage; All Organisms	Deaths; Percentage; All Organisms
Plummer, Raia and Shultz[4b]	1928-29	147	14 (9.5)*	3 (2)	3 (2)	9 (6)	19 (13)	5 (3.4)	Unclassified		7.7	17.7
	1929-30†	182	18 (9.8)	6 (3.2)	7 (3.8)	8 (4.4)	16 (8.7)	11 (6)	20 (10.9)	8 (4.4)	7.1	16.4
Present report.....	1931‡	205	27 (13)	3 (1.5)	9 (4)	16 (6)	18 (9)	1	35 (17)	18 (9)	11.5	21.9
	1932 (Jan.-July)	142	17 (12)	2 (1.4)	10 (7)	3 (2)	20 (13)	0	16 (11)	6 (4)	8	29
	1932-33	299	38 (13)	8 (2.7)	10 (3)	7 (3)	42 (14)	14 (5)	63 (21)	21 (7)	14	16
	1933-34	170	24 (14)	2 (1.2)	7 (3)	7 (3)	15 (9)	9 (5.3)	28 (16)	8 (5)	13	14
	1934-35	217	36 (16.5)	13 (6)	8 (3.6)	12 (5.5)	23 (10.5)	8 (3.6)	32 (14.7)	12 (5.5)	8.1	12.1#
Total over seven pneumonia seasons		1,362	174 (12.8)	37 (2.7)	54 (4)	62 (4.5)	153 (11.2)	48 (3.6)	194§ (14.4)	73§ (5.3)	9.6	18.3

* Figures in parentheses indicate percentages.
† Type XVIII pneumococci in 10 patients of 182 in total series.
‡ Type XXII pneumococci in 12 patients of 205 in total series.
§ Types XIV and XIX pneumococci were unclassified during 1928-1929.
# Based on deaths of 222 cases; since 5 did not have bacteriologic study at any time, they are not included elsewhere in this paper.

(Five patients with pneumonia who died in 1935 are not included here or in the entire study, since no bacteriologic study was made in these cases; they are included in table 12.) When the statistics were analyzed, however, it was found that 5.5 per cent of all patients with lobar pneumonia died as compared with 50.2 per cent of patients with bronchopneumonia (and broncholobular pneumonia).

Chart 2 illustrates a predominance of deaths in patients under 2 years of age (only dominant types are charted). Of the 186 deaths only 19 occurred in patients over 2 years. Infants from whom staphylococci and streptococci were cultured had a high mortality. Pneumococci of types VI and XIX were often associated with streptococci, as will be shown later in this paper.

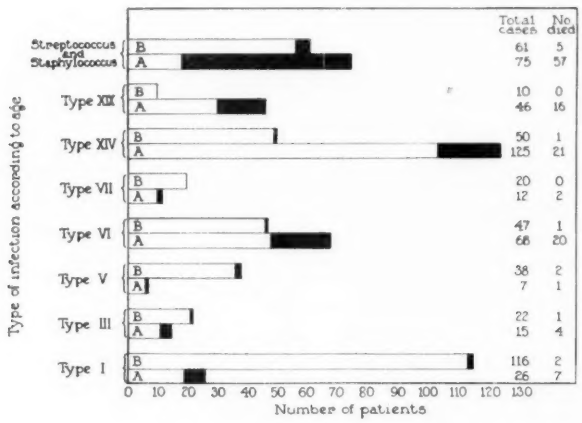

Chart 2.—Mortality rate of patients with pneumonia according to age group. (Only the most commonly encountered types of infection are charted.) A much higher mortality is shown in children under 2 years of age, regardless of infection. *A* indicates children under 2 years old; *B* indicates children from 2 to 12 years. The blocks with no shading indicate the number of patients who survived. The solid blocks indicate the number who died.

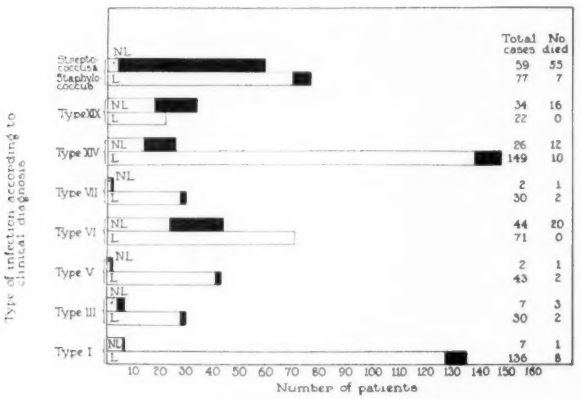

Chart 3.—Mortality rate of patients with pneumonia according to clinical type of pneumonia. (Only the predominant types of infection are charted.) A pathologic diagnosis was obtained for approximately 40 per cent of the patients who died. A much higher mortality rate was observed in the patients with nonlobar pneumonia with all types of infection, particularly the nonpneumococcic group. *L* indicates the patients with lobar pneumonia, and *NL* indicates those with nonlobar pneumonia. The blocks with no shading indicate those who survived. The blocks with solid shading indicate the number who died.

Chart 3 adds a final criterion for the outcome of pneumonia; not only are the age of the patient and the type of the infecting organism important, but these factors in a patient with bronchopneumonia make for a grave prognosis.

Over half the patients had infections associated with hemolytic streptococci and staphylococci in some degree. The high mortality noted with pneumonia due to pneumococci of types VI and XIX was most likely due to this association. Postmortem punctures of the lungs in patients dying of pneumonia caused by pneumococci of types I and XIV yielded fluid which gave pure cultures of these pneumococci as a rule.

The diagnoses given in the cases of 79 patients of the 186 who died were based on autopsy studies of sections across the whole lung. The value of sections from the whole lung in the pathologic study of pneumonia has been previously pointed out by Smith, Graef and Andrews.[16]

COMMENT

It has already become evident that diseases associated with various pneumococci differ in their clinical manifestations. A consideration of the frequency of various types of pneumococci in pneumonia, the incidence of complications and the mortality rate leads to a selection of pneumococci on the basis of relative pathogenicity. Types I, XIV, V and VII stand out as being present in a high proportion in children with lobar pneumonia and are associated with the production of empyema in a rather high proportion of cases. These types, on the other hand, are infrequently cultured from the respiratory passages of children who do not have pneumonia.

The type VI pneumococcus presents an interesting problem, being found in patients with all clinical varieties of pneumonia. Unlike types I, XIV, V and VII, it is found in a high proportion of patients not having pneumonia and rarely produces empyema. In young infants, it is associated with a fairly high mortality, with septicemia and with meningitis. Since the fatal cases are all in very young patients, a change in the host factor, rather than increased pathogenicity of the pneumococcus, is suggested as an explanation for the change in the clinical picture associated with type VI. The decreasing frequency of this organism and the serious and fatal complications associated with it with increasing age may be due to the development of immunity for this type. Since this pneumococcus is common in the first years of life, the child has probably been repeatedly exposed to type VI and may have been a carrier of this organism.

16. Smith, C. H.; Graef, I., and Andrews, E. T.: Value of Large Sections of the Lung in Pathologic Study of Pneumonia, Am. J. Dis. Child. **45**:1141 (May) 1933.

The same observations can be made of type XIX, except that meningitis and septicemia were never encountered with this organism. Also, its lower incidence in patients with pneumonia and its rarity beyond the third year suggests that there is a lower virulence for type XIX than for type VI or that children acquire immunity more rapidly for the former type. Type III has a similar story.

There is some reason to doubt the etiologic significance of pneumococci in many cases of bronchopneumonia. McCordock and Muckenfuss[17] demonstrated experimentally that a pathologic picture of bronchopneumonia in animals may be produced by a filtrable virus, with bacteria playing a secondary rôle. This finding is suggestive as a possible explanation for many cases of bronchopneumonia in the light of our clinical and bacteriologic evidence. The pneumococci found in children with bronchopneumonia, except type XIV, are those commonly found in carriers, most of them of the higher-numbered types. Gundel and Seitz,[18] in Germany, studying a large group of healthy persons, made similar observations with no special notation concerning type XIV.

Although there may be epidemics during certain years, the relative relation of the various types of pneumococci as to frequency and severity was not much altered, as is best illustrated by a study of seven pneumonia seasons in the same institution, the children's medical service of Bellevue Hospital (table 12). In 1931 a higher percentage of type XXII pneumococci was seen; in 1932-1933, an increase in the incidence of type XIV, and in 1934-1935, an increase in the incidence of type II.

Fortunately specific serum therapy in our experience is most effective against pneumonia caused by types I, XIV, V and VII, the most frequently encountered types in patients with lobar pneumonia and the types associated with disease of highest severity.

CONCLUSIONS

During four pneumonia seasons 1,033 patients with pneumonia and 425 patients having pneumococci in their respiratory passages but not having pneumonia have been studied clinically and bacteriologically.

The pneumococci most frequently found in cultures from patients with pneumonia were types I, VI and XIV. These types occurred in 41.6 per cent of the patients in this series.

The age distribution of certain pneumococci was noted. Type I was found rarely in patients under 2 years of age; type XIX, on the other

17. McCordock, H. A., and Muckenfuss, R. S.: Similarity of Virus Pneumonia in Animals to Epidemic Influenza and Interstitial Bronchopneumonia in Man, Am. J. Path. **9**:221 (March) 1933.

18. Gundel, M., and Seitz, L.: Die Pneumokokkentypen in der gesunden Bevolkerung, Klin. Wchnschr. **12**:929 (June 17) 1933.

hand, was cultured almost entirely from patients under 2 years. Types VI and XIV were more prevalent in infants and in the preschool group.

Types XIX and VI occurred most frequently (30 per cent) in the patients who did not have pneumonia. Types I and XIV were rarely obtained from this group. Pneumococci of types III, VI and XIX were found almost as commonly in patients without pneumonia as in those with it.

The pneumococci cultured from patients with bronchopneumonia were of the types commonly found in healthy carriers and, in our series, in patients without pneumonia. The type XIV pneumococcus was an exception, being present in 9.6 per cent of the patients with bronchopneumonia. Two thirds of these patients had streptococci alone or in combination with the types of pneumococci found in common carriers.

Empyema, otitis media, septicemia and meningitis were the complications observed. Many types of pneumococci were not, in our experience, associated with the development of empyema. Types I and V produced empyema in 41 patients, 39 per cent of those who had empyema, and hemolytic streptococci were obtained from 14 patients with empyema. These three organisms alone produced empyema in 55 of the 105 patients with this complication.

The mortality rate in our experience was dependent on the age of the patient, the type of infecting organism and the clinical type of pneumonia produced. The highest death rate occurred in patients under 2 years who had bronchopneumonia or broncholobular pneumonia (not lobar) and in patients infected with streptococci or staphylococci. The mortality of the entire series was 18 per cent; however, only 5.5 per cent of the patients with lobar pneumonia died, as compared with 50.2 per cent of those with bronchopneumonia.

The accurate classification of pneumococci is of great clinical significance both as to prognosis and as to treatment. Complications and mortality are definitely related to the type of infecting organism. Specific serum therapy has been most effective against pneumonia associated with selected types of pneumococci.

ACUTE APPENDICITIS IN CHILDREN

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Realization of the seriousness of acute appendicitis in children stimulates all physicians to a more determined effort at eradication of the controllable factors leading to the high mortality.

There is a reported increase in the incidence of appendicitis in childhood; however, it is probably apparent only and may doubtless be attributed in the main to greater accuracy in diagnosis, as can that reported in adults. However, the mortality rate in children with appendicitis continues to be much greater than that in patients in the higher age brackets. This may be the result of the frequent occurrence of complicating peritonitis in children. In only 27 per cent of the 250 cases recently reported by Maes and his co-workers [1] at the Louisiana State University Medical Center was the disease in the early simple acute and acute suppurative stages, and in these cases the mortality was nil. Cases in which peritonitis was a factor accounted for the death rate of 22.7 per cent in children between the ages of 2 and 6 years. It is generally known that in a majority of the patients with appendicitis entering large hospitals perforation has occurred. Analysis of these facts indicates that the improvement is in the accuracy of the final diagnosis, which is made largely by retrospection. Thus, one must conclude that the basis on which rests the hope of decreasing the mortality is diagnosis while the disease remains confined to the appendix.

The precise etiology of the symptom complex remains somewhat obscure. However, a theory which affords a simple explanation and seems to coincide most logically with the physiologic facts is one which assumes that irritation of the splanchnic nerves of the viscus and that of the somatic nerves of the parietal peritoneum are involved independently in the production of pain, tenderness and rigidity.[2] Anatomists are unable to differentiate splanchnic from somatic afferent fibers; however, physiologically and clinically the sympathetic and the cerebrospinal system differ in the nature of the stimuli capable of producing painful

1. Maes, Urban; Boyce, F. F., and McFetridge, E. M.: Acute Appendicitis in Childhood, with a Critical Analysis of Two Hundred and Fifty Cases, Surg., Gynec. & Obst. **58**:32 (Jan.) 1934.

2. Morley, John: Abdominal Pain, New York, William Wood & Company, 1931.

responses and in the character of the pain to which the two give rise.[3] Consequently an attempt has been made to interpret and evaluate the clinical manifestations of acute appendicitis on the aforementioned mechanistic basis.

In an early stage of disease of the appendix the organ may be visualized as being stimulated to peristalsis in an effort to expel its irritating contents. This activity probably is predominantly myogenic in origin. The resultant tension on the nerve endings within the wall of the viscus may prove insufficient to produce characteristic visceral pain but may be responsible for restlessness, sleeplessness, anorexia or a temporary abnormal increase in appetite, with a departure from normal intestinal habits. The accurate and indispensable history will frequently elicit the fact that one or all of these were present for an indefinite time before the onset of definite pain. Some decided mechanical interference, such as hyperplasia of the abundant lymphoid tissue in the appendix of children or exudation caused by the acute inflammatory changes, may obstruct the proximal lumen, even though this is relatively larger than that in adults. These vague symptoms change as the peristalsis becomes more exaggerated and the threshold of visceral pain is overcome. There is then evidence of intermittent, colicky pain in the upper part of the abdomen, which is at first moderate but rapidly increases in severity and is manifested in children, among other ways, by paroxysms of crying. These attacks of pain are associated with an aggravation of the nervous unrest. An increase in temperature is most unusual, and the child does not appear particularly ill. The abdomen is usually "silent" and is neither distended nor retracted. Contrary to what appears to be the general opinion, there is an absence of muscular rigidity and tenderness either in the central area where the pain is manifested or in the right iliac triangle. This lack of objective physical signs is adequately explained by considering that this phase of appendicitis is wholly concerned with the autonomic nervous system. Furthermore, acceptance of this hypothesis will lessen the tendency to temporize or to advise a hazardous purgative under the impression that the phenomena are natural and insignificant.

If the symptoms disappear as a result of the appendix having emptied its contents into the cecum, a probable diagnosis of a "gastrointestinal upset" attributed to an indiscretion in diet is strengthened. However, the tension on the autonomic nerve fibers may also be relieved by dissemination of the irritation as a result of complete interstitial inflammation or by perforation, in which case the visceral epigastric pain will gradually subside, and vomiting, if present, will cease. In

3. Hurst, A. F.: Sensibility of the Alimentary Canal, London, Oxford University Press, 1911.

this event the symptoms are followed by a decided change in demeanor, and the child is no longer in a state of jactitation but is desirous of remaining comparatively quiet in bed, frequently with the knees flexed. If the patient is old enough to respond to interrogation, there will usually be a history of superficially placed, sharp and stabbing continuous pain, usually in the right iliac fossa, which is aggravated by movement. Muscular rigidity and tenderness, both superficial and deep, make their first appearance, and a gradual rise in temperature may be expected, with a pulse rate disproportionately increased and with costal respiration. Occasionally vomiting recurs. This second phase of appendicitis represents involvement of the cerebrospinal nervous system from irritation of the parietal peritoneum by the underlying diseased viscus, the severity being in direct proportion to the degree of peritoneal involvement. If one considers the somatic pain as merely localization of the initial epigastric pain, the quality and intensity should be the same, but, on the contrary, as has been described, the second type of pain differs entirely from the first.

This syndrome of initial pain in the epigastric region and second, or localized, pain in the right iliac region has been considered typical of obstructive appendicitis. It is evidence of the marvelous physiologic coordination of the autonomic and the cerebrospinal nervous system. However, it will be seen that the completion of this composite picture must necessarily include the complication of secondary peritonitis. Furthermore, owing to the rapid course of appendicitis in children, a condition may progress from simple endo-appendicitis one day to general peritonitis the next; therefore one must see the value of making a diagnosis before somatic symptoms appear, or at least during the early stage of congestive or formative peritonitis.

It has been frequently observed that possibly only the visceral symptoms are seen, yet the excised appendix may show varying grades of inflammation, such as gangrene or gross perforation. This clinical experience is unsatisfactorily explained by the viscerosensory and visceromotor reflex theories,[4] for by this reasoning physical signs and symptoms would necessarily be present in that portion of the abdominal wall supplied by the eleventh dorsal segment, as the consensus is that the afferent splanchnic inflow of the appendix is by way of the white rami communicantes existing at this level. However, it is elucidated by considering that muscular rigidity and tenderness result only from irritation of the overlying parietal peritoneum by the diseased appendix. Therefore, when the appendix is retrocecal or retro-iliac or covered by the omentum or mesentery, these symptoms will be absent until the dis-

4. Mackenzie, J.: Symptoms and Their Interpretation, ed. 4, Shaw & Sons, Ltd., London, 1920.

ease is well advanced, since the spinal nerves of the parietes have been protected by the interposition of these structures. Likewise, when the appendix occupies a pelvic position, which is frequently the case because of the long mesocecum in a child, localized pain, muscle spasm and tenderness will not be present, since there is a lack of peripheral nerves in the abdominal wall of the segments which form the pelvis.

Most of the patients in whom the manifestations appear to be initiated by pain in the right iliac region probably have shown some if not all of the aforementioned visceral symptoms, but these were insufficient to merit attention. The exception would be cases in which the lumen of the appendix remained patent throughout the attack or in which there was some interference with the neuromuscular mechanism of the viscus, either congenital or acquired, and thus no adequate stimulus for visceral pain.

The treatment of acute appendicitis in children is immediate appendectomy, and in doubtful cases it is better to operate on the grounds of a reasonable probability of appendicitis than to wait until the diagnosis becomes clearly established.

SUMMARY

Attention has been focused on an interpretation of the early visceral symptoms of appendicitis, an accurate appreciation of which must obtain in cases in children, since the course of this disease is so rapid after the onset of somatic symptoms.

There is absence of muscular rigidity and local tenderness during the stage of endo-appendicitis; therefore, abdominal palpation is valueless except negatively.

In any case of acute abdominal disease, the possibility of appendicitis should not be excluded because of the absence of the typical symptom sequence, since this may be distorted by any of the following factors: evacuation of the irritating appendical contents into the cecum; patency of the lumen throughout the entire attack, interference with the neuromuscular mechanism of the viscus, interposition of some abdominal structure which protects the spinal nerves of the parietal peritoneum and ptosis of the appendix into the pelvis.

A tentative acceptance of the hypothesis of an independent involvement of the sympathetic and cerebrospinal nervous systems at different stages of the disease it is believed will assist in eradicating diagnostic confusion in many cases and will be conducive to earlier appendectomy, with a resulting lowered mortality rate.

902 Medical Arts Building.

RESPIRATORY METABOLISM IN INFANCY AND IN CHILDHOOD

XVII. THE DAILY HEAT PRODUCTION OF INFANTS—PREDICTIONS BASED ON THE INSENSIBLE LOSS OF WEIGHT COMPARED WITH DIRECT MEASUREMENTS

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Calorimetric studies of basal metabolism have demonstrated that the human subject from infancy to old age eliminates a constant proportion, approximately 25 per cent, of the heat produced by basal metabolism in the vaporization of water through the skin and lungs.[1] Since water vapor forms under basal conditions a relatively fixed fraction of the total insensible perspiration (insensible loss of body weight) as measured with the balance, Benedict and Root [2] used the latter as a simple and practical method for predicting the basal metabolism of adults, and Levine, Wilson and Kelly,[3] subsequently applied this method of prediction to infants in the basal state. The procedure was next extended by Newburgh and his colleagues [4] to estimations of the total heat production of adults under maintenance conditions over long periods. These workers [5] soon modified their original procedure by utilizing the water

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1. (*a*) Soderstrom, G. F., and Du Bois, E. F.: Clinical Calorimetry: XXV. The Water Elimination Through Skin and Respiratory Passages in Health and Disease, Arch. Int. Med. **19**:931 (May) 1917. (*b*) Levine, S. Z., and Wilson, J. R.: Respiratory Metabolism in Infancy and in Childhood: IV. Elimination of Water Through the Skin and Respiratory Passages of Children, Am. J. Dis. Child. **33**:204 (Feb.) 1927; (*c*) VII. Elimination of Water Through the Skin and Respiratory Passages of Infants, ibid. **35**:54 (Jan.) 1928.

2. Benedict, F. G., and Root, H. F.: Insensible Perspiration: Its Relation to Human Physiology and Pathology, Arch. Int. Med. **38**:1 (July) 1926.

3. Levine, S. Z.; Wilson, J. R., and Kelly, M.: Prediction of the Basal Metabolism of Infants from the Measured Insensible Perspiration, Proc. Soc. Exper. Biol. & Med. **25**:554, 1928.

4. Johnston, M. W., and Newburgh, L. H.: The Determination of the Total Heat Eliminated by the Human Being, J. Clin. Investigation **8**:147, 1930.

5. Newburgh, L. H.; Wiley, F. H., and Lashmet, F. H.: A Method for the Determination of Heat Production Over Long Periods of Time, J. Clin. Investigation **10**:703, 1931.

vapor alone (H_2O) rather than the total insensible loss ($H_2O + [CO_2 - O_2]$) since $CO_2 - O_2$ varies with the composition of the metabolic mixture (respiratory quotient) and is independent of the level of heat production. Lavietes [6] recently reviewed the evidence and concluded that the method, even after revision, yielded inconstant results except under well defined experimental conditions.

The validity of employing the insensible perspiration or, more accurately, its component, water vapor, as a means of estimating the total heat production over long periods has heretofore not been subjected to the direct test of concurrent twenty-four hour measurements of heat production by the method of indirect calorimetry and of insensible perspiration by the method of weighing.

The infant is a particularly suitable subject for such a study because (1) prolonged and concurrent measurements of heat production and of insensible loss of weight may be made under conditions more closely approximating the normal environment than is possible with adults and (2) extraneous factors, such as diet and activity, which are known to influence both mechanisms, tend to vary more in the ordinary adult environment, and these spontaneous variations are less subject to control in adults than in infants, who receive simple milk formulas and spend their entire day in bed. This paper reports measurements with a balance of the twenty-four hour insensible loss of weight of three healthy infants in eight periods of from three to five days each. On the earlier days of the periods of observation, the infants lived under conditions simulating the normal infantile environment; on the final day of each period, they were confined in a respiration chamber, and their energy exchange was determined simultaneously with their insensible loss of weight.

MATERIAL AND METHODS

The clinical histories of the infants and a detailed description of the methods employed to measure the excreta in the form of urine and feces, the twenty-four hour insensible loss of moisture from the skin and the lungs and the heat production were given in an earlier communication.[7] The infants resided in a special metabolism ward with an adjoining diet kitchen [8] throughout the periods

6. Lavietes, P. H.: The Metabolic Measurement of Water Exchange, J. Clin. Investigation **14**:57, 1935.

7. Levine, S. Z.; McEachern, T. H.; Wheatley, M. A.; Marples, E., and Kelly, M.: The Respiratory Metabolism in Infancy and in Childhood: XV. Daily Energy Requirements of Normal Infants, Am. J. Dis. Child. **50**:596 (Sept.) 1935.

8. Levine, S. Z., and Wilson, J. R.: The Respiratory Metabolism in Infancy and in Childhood: I. Basal Metabolism of Children, Am. J. Dis. Child. **31**:323 (March) 1926. Wilson, J. R.; Levine, S. Z., and Rivkin, H.: The Respiratory Metabolism in Infancy and in Childhood: II. Ketosis and

(Footnote continued on next page)

of observation. They received constant diets consisting of powdered whole milk, cane sugar and water for at least three days and usually more prior to the onset of experimental periods. Feedings were at four hour intervals, the one at 2 a. m. being omitted in all except the first observation (experiment 166). The twenty-four hour insensible loss of weight of the infants was measured with the balance on each of the three to five days of each period, and the energy exchange was simultaneously measured in the respiration chamber throughout from twenty and one-half to twenty-two hours of the last day of the period. In one observation (experiment 166) approximately one month intervened between measurements of the insensible loss while the infant was outside and while he was inside the respiration chamber; in another (experiment 167) this measurement was made only on the day of the calorimetric observation.

The following data were required for the twofold purpose of predicting the total heat production from the insensible loss of water and of directly assessing the reliability of these predictions with direct measurements:

Total Weight and Carbohydrate Content of the Diet.—The daily intake was weighed with an accuracy of 0.1 Gm. All weighing was done by the same person. Besides carbohydrate,[9] analyses of the two other organic foodstuffs, protein (macro-Kjeldahl method) and fat,[10] were made in duplicate on thoroughly mixed pooled samples of the powdered milk. The results obtained by chemical analysis closely paralleled the average figures submitted by the manufacturer, and the caloric equivalents corresponded closely to the values determined with the bomb calorimeter.

Total Weight and Nitrogen Content of the Urine and Feces.—The weights were measured with an accuracy of 0.1 Gm. The weight in grams of the urinary nitrogen (macro-Kjeldahl method) multiplied by the constants 6.25 and 25.63 (6.25×4.1) afforded a measure of the protein metabolism in grams and calories, respectively. The amount of urinary nitrogen rather than the total amount of excretory nitrogen was used since, according to Rubner and Lusk,[11] the physiologic caloric equivalent, 4.1 per gram of protein, automatically covers the waste heat excreted as nitrogenous end-products in the feces. Mitchell and Hamilton[12] concluded from extensive researches on the subject that "the so-called metabolic nitrogen of the feces consists predominantly of nitrogenous waste products of

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the Respiratory Exchange in Children, ibid. **31**:335 (March) 1926. Levine, S. Z.; Wilson, J. R.; Berliner, F., and Rivkin, H.: The Respiratory Metabolism in Infancy and in Childhood: VI. The Specific Dynamic Action of Food in Normal Infants, ibid. **33**:722 (May) 1927.

9. Folin, O., and Wu, H.: A System of Blood Analysis, J. Biol. Chem. **38**:106, 1919; A Simplified and Improved Method for the Determination of Sugar, ibid. **41**:367, 1920.

10. Association of Official Agricultural Chemists: Official and Tentative Methods of Analysis Compiled by the Committee on Editing Methods of Analysis, ed. 3, Washington, D. C., Association of Official Agricultural Chemists, 1930, p. 217.

11. Rubner and Lusk, cited by Du Bois, E. F.: Basal Metabolism in Health and Disease, ed. 2, Philadelphia, Lea & Febiger, 1927, p. 40. Lusk, G.: The Science of Nutrition, ed. 4, Philadelphia, W. B. Saunders Company, 1928, p. 39.

12. Mitchell, H. H., and Hamilton, T. S.: The Biochemistry of the Amino Acids, American Chemical Society Monograph Series, New York, The Chemical Catalog Company, 1929, p. 239.

digestion rather than of metabolism." It may be noted parenthetically that for the comparative purposes of this paper either of the two calculations could have been adopted without affecting the interpretation of the results presented later.

Changes in Body Weight and Total Insensible Perspiration.—The infants were weighed daily with a delicate balance having a capacity of 10 Kg. and an accuracy of 0.05 Gm.[13] The weight of the gaseous emanations leaving the body by way of the skin and lungs was determined for each twenty-four hours of the periods of observation by subtracting from the initial body weight plus the weight of the daily intake the final body weight plus the weight of the daily output of urine and feces.

Respiratory Metabolism.—The twenty-four hour heat production and the total gaseous exchange, including the elimination of carbon dioxide, consumption of oxygen and vaporization of water, were determined by direct measurement in a closed circuit respiration chamber of the Benedict-Talbot type on the final day of each period. The apparatus, technic of measurements and methods of calculation were fully described in earlier papers.[8] The infants spent from twenty and one-half to twenty-two hours of the day and night in the respiration chamber. The remaining two to three and one-half hours were occupied in the removing of the infant from the chamber, feeding him, recording the rectal temperature, reinserting him in the chamber and allowing the environmental temperature and humidity to establish equilibrium.

The behavior of the infants throughout the three to five days of the experimental periods, including the final days, both while inside and while outside the respiration chamber, was observed by inspection, recorded from minute to minute for each twenty-four hours and partitioned into periods of sleep and wakefulness. This record of activity, while admittedly not ideal, provided acceptable data for expanding the energy exchange of the infants, as directly measured for from twenty and one-half to twenty-two hours of the final day of each period while the child was asleep and while he was awake, in terms of the twenty-four hours of each day of the period.

The environmental temperature and relative humidity were recorded at four hour intervals throughout the periods of observation. The measurements of temperature were taken with an accurate mercury thermometer; those of relative humidity, with a psychrometer and a hair hygrometer while the infants were outside and inside the respiration chamber, respectively. All measurements of air conditions were made in as close proximity to the infants as possible.

The concurrent measurements of insensible loss and of the respiratory metabolism permitted a direct comparison of the twenty-four hour heat production, estimated from the insensible loss according to the method of Newburgh[14] and obtained by actual measurement in the respiration chamber.

The data also made possible a similar comparison of the twenty-four hour heat production, predicted as heretofore from the measured insensible loss on the earlier days of the period and calculated from the calorimetric data, allowance being made for differences in activity while the infant was outside the respiration chamber.

It must be emphasized that these studies were made on infants and consequently the results, as reported, are not necessarily applicable to adults.

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13. Levine, S. Z.; Wilson, J. R., and Kelly, M.: The Insensible Perspiration in Infancy and in Childhood: I. Its Constancy in Infants Under Standard Conditions and the Effect of Various Physiologic Factors, Am. J. Dis. Child. **37**:791 (April) 1929.

14. Newburgh, L. H., and MacKinnon, F.: The Practice of Dietetics, New York, The Macmillan Company, 1934, chap. 5. Newburgh, Wiley and Lashmet.[5]

OBSERVATIONS

Prediction of Twenty-Four Hour Heat Production from the Insensible Loss.—According to the method advocated by Newburgh, for use with adults, the factors which must be known in order to estimate the heat production are: (1) the insensible loss of body weight (insensible perspiration), (2) the amount of dietary carbohydrate and (3) the amount of urinary nitrogen. The reasons for employing the amount of urinary nitrogen rather than the total amount of excretory nitrogen in these studies have already been stated. These data for the last days of the eight periods of observation are given in table 1.

From these data, the composition of the metabolic mixture may be determined if the following conditions are fulfilled: (1) All the carbohydrate of the diet is burned in the body; (2) the urinary nitrogen

TABLE 1.—*Data Required to Estimate the Twenty-Four Hour Heat Production from the Insensible Perspiration*

Subject	Age, Months	Weight, Kg.	Experiment	Date	Insensible Perspiration Balance, Gm. per 24 Hours	Carbohydrate* in Daily Diet, Gm.	Nitrogen in Urine, Gm. per 24 Hours	Type of Diet
H. S.	7⅓	7.54	166	11/16/31	358	72.2	3.9276	Maintenance
H. S.	8	8.00	167	12/ 4/31	341	73.9	4.0322	Maintenance
J. F.	9¼	7.29	169	1/ 8/32	386	77.8	4.4415	Maintenance
J. C.	4½	6.35	170	2/19/32	272	59.1	3.3967	Maintenance
J. C.	4¾	6.06	171	2/26/32	180	29.6	2.2227	Low caloric
J. C.	5	6.71	172	3/ 4/32	308	75.1	4.4679	High caloric
J. C.	6½	7.39	173	4/15/32	293	58.9	3.5838	Maintenance
J. C.	6¾	7.24	174	4/21/32	287	58.9	4.3219	Low fluid

* The carbohydrate of the diet consisted of lactose in powdered milk and added sucrose. The figures in this column represent the results of chemical analysis.[9] They closely approximate the average figures submitted by the manufacturer and the caloric equivalents obtained with the bomb calorimeter.

(× 6.25) is an accurate measure of the protein metabolism, and (3) a constant proportion, 24 per cent, of the total heat produced leaves the body in the vaporization of water (insensible loss of water × 0.584 = total calories × 0.24). Peters and his co-workers [15] devised a simple equation for calculating the amount of fat in the metabolic mixture ($F = \frac{I\,L - 2.2\,C. - 1.69\,P}{3.78}$, in which F represents the amount of fat in the metabolic mixture, I L the total insensible loss of weight measured with the balance, C the amount of dietary carbohydrate and P the amount of excretory nitrogen × 6.25). This equation was accepted by Newburgh as a valid condensation of his calculations.[14] The heat production is computed from the caloric equivalents of the organic materials metabolized in the body. Table 2 presents a summary of these

15. Peters, J. P.; Kydd, D. M., and Lavietes, P. H.: A Note on the Calculation of Water Exchange, J. Clin. Investigation **12**:689 (July) 1933.

calculations, the last column giving the figures for the twenty-four hour heat production of the infants, calculated in this manner for the final day of each experimental period.

Comparison of Measured and Predicted Twenty-Four Hour Heat Production.—The energy exchange of the infants directly measured by indirect calorimetry is compared in table 3 with the results predicted from their insensible loss of water. In this table, the number of calories actually measured in the respiration chamber for from twenty and one-half to twenty-two hours was expanded to that for the full duration of from twenty-four to twenty-five hours (table 4) by assuming that during their sojourn of from two to three and one-half hours outside the chamber the infants were producing heat at the

TABLE 2.—*Calculation of the Composition of the Metabolic Mixture and Heat Production (from Data in Table 1)*

Experiment	Carbohydrate (in Diet), Gm. per 24 Hours	Protein (Urine Nitrogen × 6.25), Gm. per 24 Hours	Fat* $\left(\frac{IL - 2.10\,C - 1.77\,P}{3.76}\right)$, Gm. per 24 Hours	Calories† per 24 Hours
166	72.2	25.4	43.0	800
167	73.9	26.1	37.2	756
169	77.8	28.7	45.6	861
170	59.1	22.0	29.0	602
171	29.6	14.4	24.5	408
172	75.1	28.9	26.4	672
173	58.9	23.2	34.1	654
174	58.9	27.0	31.0	640

* The equation adopted for calculating the fat in the metabolic mixture differs slightly from those of Peters[15] $\left(F = \frac{IL - 2.12\,C - 1.69\,P}{3.78}\right)$ and of Newburgh[14] $\left(F = \frac{IL - 2.06\,C - 1.73\,P}{3.63}\right)$.

† The customary constants were used: P × 4.1; F × 9.3; C × 4.1.

same rate as their maximum rate of production of heat while they were awake within the chamber. The validity of this assumption will be considered in a later section. The detailed calorimetric data and the results of alcohol checks, which were made periodically to control the reliability of the observations, appeared in a previous paper.[7]

In every instance the twenty-four hour heat production as estimated from the insensible loss of water exceeded the measured energy exchange, the discrepancies ranging between +3 and + 46 per cent and exceeding + 20 per cent in seven of the eight observations. It is significant that in the one observation in which the discrepancy was minimal (experiment 171) the infant was receiving a submaintenance diet with a caloric content sufficient only to cover his measured basal energy requirements.

The frequency and magnitude of the observed deviations justify the conviction that the method proposed for predicting the total heat pro-

duction of adults from their measured insensible loss of water (or total insensible perspiration) is not applicable to infants in prolonged calorimetric studies conducted under maintenance (as opposed to basal) conditions.

It becomes necessary in attempting to explain the divergent results to investigate the possible sources of error in the technic of the measure-

TABLE 3.—*Comparison of Measured and Calculated Twenty-Four Hour Heat Production*

Experiment	Calories per 24 Hours		Deviation	
	Measured in Chamber	Estimated from Insensible Loss	Calories	Percentage
166	555	800	245	+44
167	576	756	180	+31
169	591	861	270	+46
170	460	602	142	+31
171	395	408	13	+ 3
172	510	672	162	+32
173	510	654	144	+28
174	530	640	110	+21

TABLE 4.—*Partition of Heat Production While the Infants Were Inside (Measured) and While They Were Outside (Calculated) the Respiration Chamber*

Experiment (1)	Inside			Outside*			Total			Calories, per Hour		
	Duration (2)		Calories (3)	Duration (4)		Calories (5)	Duration (6)		Calories (7)			
	Hrs.	Min.		Hrs.	Min.		Hrs.	Min.		Basal‡	Outside Chamber†	Rise, Percentage
166	21	39	463	3	14	92	24	53	555	19.8	28.5	44
167	21	57	502	2	39	74	24	36	576	21.3	27.9	31
169	22	5	509	2	55	82	25	0	591	20.5	28.1	37
170	20	23	372	3	37	88	24	0	460	17.2	24.3	41
171	20	58	335	3	2	60	24	0	395	14.4	19.8	37
172	20	44	426	3	16	84	24	0	510	19.1	25.7	35
173	20	25	419	3	35	91	24	0	510	18.4	25.4	38
174	21	10	459	2	50	71	24	0	530	20.9	25.1	20

* The number of calories shown in this column were calculated on the basis of a metabolic rate assumed to correspond with the heat production of the infant directly measured while awake within the respiration chamber.

† Derived from columns 4 and 5.

‡ The basal calories represent the average numbers per hour measured while the infants were asleep in the respiration chamber.

ment of heat production and of insensible perspiration and to assess the credibility of the assumptions embodied in the calculations. Such an appraisal necessitates examination of the following factors: (1) the method employed to estimate the energy exchange of the infants for the two to three and one-half hours spent outside the respiration chamber, (2) measurements of the insensible perspiration with the balance, (3) employment of the amount of urinary nitrogen as a measure of the protein metabolism, (4) adoption of the amount of dietary carbo-

hydrate as an index of the amounts metabolized, (5) the equation for calculating the amount of fat in the metabolic mixture, (6) the statement that under maintenance conditions 24 per cent of the total heat produced is removed from the body in the vaporization of water from the skin and lungs and (7) the further implication that the assumed percentage represents a constant and unchanging fraction of the heat produced from day to day. Each of the aforementioned possibilities, with particular reference to their applicability to infants, will be separately considered.

Estimation of Heat Production While Outside the Respiration Chamber.—The reliability of measurements of the heat production of the infants while inside the respiration chamber was attested by the consistent results obtained in the alcohol checks, previously reported.[7] Since, however, the insensible perspiration was measured with the balance for the entire day (from twenty-four to twenty-five hours) and the heat production only while the infant was in the respiration chamber (from twenty and one-half to twenty-two hours), it became necessary to assign a certain number of calories for the two to three and one-half hours spent outside the chamber in order to correlate the two measurements on the same time basis. The number of calories was allotted on the assumption that the heat production of the infants while outside the respiration chamber corresponded per unit of time with their metabolic rates measured while they were awake within the chamber.

The metabolic increments of from sixty to ninety-two calories allocated in this manner for the 11 to 15 per cent of the day spent outside the respiration chamber represented increases of from 20 to 44 per cent above the respective basal levels of the infants as measured while they were asleep within the chamber (table 4). The energy expenditure, as estimated, is probably not far from the true caloric output, since in none of the observations were the infants restless nor did they cry during their relatively brief residence outside the chamber.

The possibility exists, however, that the freedom from artificial restraint, the acts of eating and having the rectal temperature taken and the unavoidable absence of measurements immediately following meals while the infants were outside the respiration chamber might have resulted in augmentation of heat production (unmeasurable with present methods) in excess of the measured metabolic rate while they were awake within the chamber—the standard adopted in our calculations. To meet this criticism, a hypothetical elevation in heat production of 100 per cent (instead of the allotted increase of from 20 to 44 per cent) above sleeping levels was allowed for the relatively minor degrees of activity while the infants were outside the chamber. This assumed increment of metabolic increase notably exceeds the

increments previously reported for equivalent or even greater degrees of activity in calorimetric studies of infants.[16] The differences between the twenty-four hour heat production of the infants predicted from the insensible loss of water and that calculated from the calorimetric data on this assumed basis remained at the significantly high level of from -3 to $+36$ per cent and exceeded 20 per cent in five of the eight observations, in contrast to the deviations of from $+3$ to $+46$ per cent shown in table 3.

The conclusion seems warranted that the error, if any, in the method for arriving at an estimate of the caloric output of the infants during their brief sojourns outside the respiration chamber played no rôle or at most a trivial one in explaining the discrepancies between their predicted and measured twenty-four hour heat production.

The Reliability of Measurements of the Insensible Perspiration with the Balance.—This measurement represents the gaseous emanations which leave the body by way of the skin and lungs and which consist essentially of water vapor (in the absence of sweating) plus carbon dioxide less oxygen ($H_2O + CO_2 - O_2$). The calorimetric data, which included direct measurements of each of these components, afforded a means of checking the reliability of measurements of the total insensible perspiration which had been made with the balance.[7]

The similarity of the figures for the twenty-four hour insensible perspiration secured in toto with the balance and by algebraic summation of the results of calorimetric measurements while the patient was in the respiration chamber ($H_2O + CO_2 - O_2$) furnishes striking evidence of the technical reliability of the former measurements. When one recalls the diverse technics employed in the two sets of measurements, one is impressed by the narrow range of deviation shown in table 5. Technical errors in measurement of the insensible loss of moisture with the balance may therefore be excluded as an important factor in explaining aberrations in the predicted twenty-four hour heat production of the infants observed.

The similarity of the two series of figures also serves to affirm the validity of the previously described method for estimating the caloric output for the two to three and one-half hours spent outside the respiration chamber since the methods for expansion of the individual components of the insensible loss and of heat production from the measured periods of from twenty and one-half to twenty-two hours to total days was the same in both instances.

16. Talbot, F. B.: Twenty-Four Hour Metabolism of Two Normal Infants, with Special Reference to the Total Energy Requirements of Infants, Am. J. Dis. Child. **14**:25 (July) 1917. Wilson, J. R.; Levine, S. Z., and Kelly, M.: The Respiratory Metabolism in Infancy and in Childhood: XI. The Respiratory Exchange in Marasmus; Effect of Muscular Activity, ibid. **39**:736 (April) 1930.

The Amount of Urinary Nitrogen as a Measure of the Protein Metabolism.—In short observations of several hours, there may be a sufficient lag in the urinary excretion of nitrogen to cause an appreciable error in estimation of the amount of protein metabolized during the period. This source of error is reduced by the prolongation of experi-

TABLE 5.—*Comparison of Twenty-Four Hour Insensible Perspiration Measured by Calorimetry and with the Balance*

	Calorimetry				Balance	Deviation	
Experiment	Water, Gm.	Carbon Dioxide, Gm.	Oxygen, Gm.	Insensible Perspiration ($H_2O + CO_2 - O_2$), Gm.	Insensible Perspiration, Gm.	Gm.	Percentage
166	333	198	164	367	358	9	+ 3
167	295	206	170	330	341	11	— 3
169	402	213	174	441	386	55	+14
171	236	165	136	264	272	8	— 3
171	190	132	119	202	180	22	+12
172	279	187	150	316	308	8	+ 3
173	233	178	152	260	293	33	—11
174	282	187	158	311	287	24	+ 8
Average							+ 2.7

TABLE 6.—*Excretion of Nitrogen in Urine on Individual Days of Periods* *

	Urinary Nitrogen per 24 Hours, Gm.						Deviation, Gm.	
	Days Outside Respiration Chamber					Calorimeter Specimen		
Experiment	1	2	3	4	Average		Nitrogen	Protein (N × 6.25)
166†	4.029	3.897	4.231	4.153	4.0775	3.9276	0.1499	0.9
167‡	.....	.....	.....	.....	.....	4.0322	0.0453	0.3
169	4.4943	4.3234	4.5712	.....	4.4630	4.4415	0.0215	0.1
170	3.5917	3.4612	3.4769	.....	3.5150	3.3967	0.1183	0.7
171	2.3260	2.3630	2.3371	.....	2.3420	2.2227	0.1193	0.7
172	4.9483	4.8280	4.9742	.....	4.8165	4.4679	0.3486	2.2
173	3.7097	3.7239	3.7060	.....	3.7132	3.5838	0.1294	0.8
174	4.6355	4.2097	.....	.....	4.3637	4.3219	0.0418	0.3

* Preformed protein (albumin) and keto-acids were absent from all the specimens.
† In this observation approximately one month intervened between measurements on the days preceding and the day of the calorimetric study.
‡ The urinary nitrogen was not determined on the days preceding this calorimetric observation, but the average figure in experiment 166 (4.0775 Gm.) is suitable for comparison since the diets in the two experiments on this infant were identical.

mental periods with the subject on a constant dietary regimen as in these studies. The amount of urinary nitrogen on the days immediately preceding calorimetric observations is compared in table 6 with the nitrogen content of the calorimetric specimens.

The constancy of the daily figures demonstrates that the infants were in dietary equilibrium and establishes the validity of employing the amount of urinary nitrogen as an index of the protein metabolism in these observations. The average values for the two to four earlier days of the period differed from the values for the final day when

calorimetric measurements were made by less than 1 Gm. in terms of protein metabolized per twenty-four hours in all except one instance (experiment 172). Even in the latter, the deviation of 2.2 Gm. (or 9 calories) resulted in an error of less than 2 per cent in the calculation of the total heat production in a twenty-four hour period (510 calories). The error from this source in the remaining observations was always less than 1 per cent.

Since calculation of the energy metabolism from the insensible loss of water and that by indirect calorimetry are based on similar use of the amount of urinary or of total excretory nitrogen, this factor is excluded a priori from explanation of the observed discrepancies between the predicted and the measured heat production of the infants. The constant results obtained on individual days do, however, serve to control the reliability of the reported figures for protein metabolism.

The Amount of Carbohydrate in the Diet and in the Metabolic Mixture.—As previously noted, one of the assumptions which determines the validity of estimations of heat production from the insensible loss of water is that the amount of carbohydrate of the diet under conditions of dietary equilibrium is an accurate measure of the amount of carbohydrate in the metabolic mixture. This assumption is based on the well recognized dictum that when adequate amounts of carbohydrate are available the human subject preferentially burns it to the relative exclusion of the other two organic foodstuffs, protein and fat. Experimental verification of this generally accepted but hitherto unproved dictum as it applies to infants is furnished by the data in table 7, in which the amount of dietary carbohydrate determined by chemical analysis is compared with the amount of metabolized carbohydrate determined by the method of indirect calorimetry.

The relatively trivial differences of from 0.2 to 5.4 Gm. between the amount of carbohydrate in the daily diet and that in the twenty-four hour metabolic mixture, irrespective of the level of intake (from 29.6 to 77.8 Gm.), provide experimental proof that infants as well as adults are essentially "carbohydrate burners." In consequence, the procedure of employing the amount of dietary carbohydrate under stabilized conditions of diet as a measure of metabolized carbohydrate is applicable to these subjects. The maximum error from this source was 4 per cent in experiment 169 (5.4 Gm., or 22 calories, in a total daily output of 591 calories) and was less than 2 per cent in all the remaining observations.

Calculation of the Amount of Fat in the Metabolic Mixture.—Experimental evidence has been presented in the preceding sections that in infants, as in adults, the amount of protein and of carbohydrate oxidized in the body may be accurately estimated under fixed condi-

tions of diet from the amount of urinary nitrogen and the amount of dietary carbohydrate. It now remains to appraise the method advocated for adults for estimating the amount of fat in the metabolic mixture and to investigate its applicability to infants. The constants employed and the derivation of the equations adopted by both Peters and Newburgh for this purpose have been fully considered in the articles published by them and their associates.[17] The equations are formulated on the assumption that with an unchanging body tempera-

TABLE 7.—*Comparison of the Amount of Carbohydrate in the Daily Diet and That in the Twenty-Four Hour Metabolic Mixture*

Experiment	Carbohydrate per 24 Hours, Gm.		Deviation, Gm.
	In Diet	Metabolized	
166	72.2	73.1	0.9
167	73.9	76.2	2.3
169	77.8	83.2	5.4
170	59.1	61.2	2.1
171	29.6	29.4	0.2
172	75.1	76.2	1.1
173	58.9	57.5	1.4
174	58.9	61.6	2.7

TABLE 8.—*Comparison of the Amount of Fat in the Metabolic Mixture Determined by Direct Measurement and by Calculation from Equation* $\left(F = \frac{\text{I L} - 2.1\ \text{C} - 1.77\ \text{P}}{3.76}\right)$

Experiment	Fat Metabolized per 24 Hours, Gm.		Deviation		
	Measured	Calculated	Gm.	Calories	Percentage
166	16.1	43.0	26.9	250	167
167	16.9	37.2	20.3	189	120
169	14.2	45.6	31.4	292	221
170	12.8	29.0	16.2	151	127
171	23.2	24.5	1.3	12	6
172	8.6	26.4	17.8	166	207
173	19.3	34.1	14.8	138	77
174	17.5	31.0	13.5	126	77

ture the amount of heat dissipated in the vaporization of water (0.584 calories per gram under ordinary environmental conditions) represents exactly 24 per cent of the total heat production. The amount of fat catabolized may therefore be readily determined by subtracting from the total number of calories, thus obtained, the number of calories derived from the oxidation of carbohydrate and protein. The twenty-four hour figures for metabolized fat, calculated from the equation and directly determined by calorimetric measurement, are given in table 8.

The enormous differences between the two series of figures, which were found in all except one observation on the basal dietary regimen

17. Newburgh and Mackinnon.[14] Peters and others.[15]

(experiment 171), show the fallacy of routine application to infants of the equation adopted for adults. Differences between the measured and the calculated number of calories derived from the combustion of fat in the seven observations of infants on maintenance dietary regimens ranged from 126 to 292 calories per day, or from 77 to 221 per cent. According to the equation, the number of calories derived from fat is obtained by deducting from the total number of calories as predicted from the insensible loss of water the number contributed by the oxidation of carbohydrate and protein. Since it has been shown that in these observations the methods for computing the number of calories derived from carbohydrate and from protein were valid, the obvious inference is that the sole factor which invalidated predictions of the twenty-four hour maintenance metabolism of the infants from the amount of water vaporized was the use of the constant, 24 per cent, which had proved reliable for the estimation of the metabolism of adults in the basal state.

Relation of the Heat Loss in the Vaporization of Water to Total Heat Production.—In basal calorimetric observations a similar relation between the total heat production and the heat loss in the vaporization of water has been shown to exist in infants as in adults, the proportion of heat lost in this manner by normal infants averaging 27 per cent and falling within the extreme limits of 23 and 30 per cent.[1e] The results of this investigation are consistent with the previously published figures, the average values for the proportion of heat lost in the vaporization of water in the basal (sleeping) periods of from twelve to fifteen hours falling within the established normal range in six of the eight observations and averaging 29.7 per cent for all observations (table 9). In the two periods of observation (experiments 166 and 169) in which larger fractions of the heat produced by basal metabolism were lost in vaporization (35 and 38 per cent), the relative humidity of the air in the respiration chamber was exceptionally high, averaging 53 and 60 per cent and attaining maximum levels of 86 and 88 per cent, respectively (table 10). If these two experiments are excluded, the average percentage of heat lost in vaporization in the periods of sleep of the remaining six observations was 27.4 per cent, the counterpart of the average figure previously found in the basal calorimetric measurements during short periods.[1e]

The relationship observed in the periods of sleep between the production of heat and the loss of heat in the vaporization of water was not maintained at the same levels in the periods of calorimetric measurement of from six to ten hours during which the infants were awake. In these periods the proportion of heat lost in vaporization varied between 27 and 41 per cent in individual observations, exceeded in six of the eight periods of observation the upper limit of 30 per cent pre-

viously found under basal conditions and averaged 33.8 per cent for all periods. In only one period of observation (experiment 173) was the average percentage of heat lost in vaporization lower in the periods

TABLE 9.—*Proportions of the Total Amount of Heat Produced Dissipated in the Vaporization of Water (Sleeping, Wakeful and Total Periods)*

	Sleeping Periods						Wakeful Periods*						Total Periods							
		Duration						Duration						Duration		Percentage of Total Period				
Experiment	Number of Periods	Hours	Minutes	Heat, Calories	Water, Gm.	Calories Lost in Vaporization, Percentage	Number of Periods	Hours	Minutes	Heat, Calories	Water, Gm.	Calories Lost in Vaporization, Percentage	Number of Periods	Hours	Minutes	Awake Restless and Crying	Heat, Calories	Water, Gm.	Calories Lost in Vaporization, Percentage
166	11	12	44	252	152	35.2	6	8	55	211	129	35.7	17	21	39	22	14	463	281	35.4
167	9	12	0	256	120	27.4	8	9	57	246	131	31.1	17	21	57	22	7	502	251	29.2
169	9	13	30	277	182	38.4	6	8	35	232	163	41.0	15	22	5	35	23	509	345	39.6
170	8	14	15	244	97	23.2	7	6	8	128	86	39.2	15	20	23	19	9	372	182	28.6
171	5	12	5	174	82	27.5	7	8	53	161	78	28.3	12	20	58	31	3	335	161	28.1
172	6	15	15	291	151	30.3	6	5	29	128	78	35.6	12	20	44	21	7	426	229	31.4
173	7	11	0	202	93	26.9	6	9	25	217	99	26.6	13	20	25	33	4	419	192	26.8
174	5	12	25	257	126	28.6	6	8	45	202	113	32.7	11	21	10	23	0	459	238	30.3
Average.....						29.7						33.8								31.2

* The infants did not remain awake throughout the entire duration of these periods, since the latter were terminated only after the infants fell asleep and remained asleep for at least fifteen minutes in order to allow the temperature and humidity to establish equilibrium.

TABLE 10.—*Relation of Environmental Conditions to Percentile Elimination of Heat in Vaporization During Sleeping and Active Periods (Data Arranged in Ascending Fashion According to Percentages Lost in the Sleeping Periods)*

Experiment	Average Temperature, C.		Average Relative Humidity		Percentage of Calories Vaporized	
	Asleep	Awake	Asleep, Percentage	Awake, Percentage	Asleep	Awake
170	23.4 (22.6 to 24.1)	23.7 (22.8 to 24.4)	32 (29 to 36)	41 (35 to 55)	23.2	39.2
173	23.8 (23.5 to 24.3)	24.3 (23.7 to 24.4)	32 (24 to 45)	41 (32 to 49)	26.9	26.6
167	25.5 (25.0 to 26.0)	25.2 (24.0 to 25.8)	41 (33 to 51)	54 (36 to 67)	27.4	31.1
171	23.9 (23.4 to 24.2)	24.2 (23.2 to 25.1)	32 (25 to 38)	44 (29 to 60)	27.5	28.3
174	26.9 (26.5 to 27.3)	26.9 (26.5 to 27.2)	39 (37 to 42)	49 (47 to 54)	28.6	32.7
172	24.3 (23.9 to 24.8)	23.6 (22.9 to 24.2)	34 (32 to 37)	46 (40 to 51)	30.3	35.6
166	26.2 (25.5 to 27.6)	26.2 (25.4 to 27.3)	53 (41 to 86)	62 (40 to 84)	35.2	35.7
169	24.1 (23.5 to 25.1)	24.5 (23.5 to 25.4)	60 (41 to 88)	67 (55 to 81)	38.4	41.0
Average	24.8	24.8	40	51	29.7	33.8

of activity than in the periods of sleep, and even in that instance the difference was negligible (26.6 as against 26.9 per cent).

That the higher losses of heat in vaporization in the periods of wakefulness were dependent more on the activity of the infants per se than on coincidental fluctuations of environmental temperature and humidity is suggested by the data presented in table 10. In this table, the periods of sleep and those of activity are again segregated, and the

data are arranged in ascending fashion according to the percentage of calories dissipated in vaporization in the former periods. Within the range of temperature present in these observations, no correlation is evident between this factor and the percentile elimination of heat in vaporization either in the basal periods or in the periods of activity.

The effect of environmental relative humidity would appear at first glance to be of greater importance. As previously noted, the basal heat loss in vaporization attained its maximum levels in the periods of sleep of the two observations in which the relative humidity was highest (experiments 166 and 169). Further, it will be noted that the relative humidity was always higher in the period of activity of each observation at the time when the loss of heat in vaporization reached its maximum level (table 10). The results obtained in the basal periods suggest that a critical level of relative humidity may be present, above which an abrupt and sudden acceleration in the output of moisture from the skin and lungs occurs. The alternative possibility presents itself that at inordinately high levels of relative humidity moisture is deposited on the walls and furnishings of the respiration chamber, which may be vaporized during the course of the observations, thus leading to spurious accretions in the amount actually measured as water vaporized. The correlation between the higher levels of relative humidity and the augmented heat losses in vaporization found in the periods of activity may again be an accidental one, attributable more to technical factors of measurement than to a causal relationship. In periods of increased activity the ventilating system of the respiration chamber may fail to blow off the additional moisture eliminated by the infants as a consequence of their activity, with the result that water accumulates on the walls and contents of the chamber and leads to an elevation in the relative humidity of the surrounding air. If this should occur, part of the water vaporized by the infants in the periods of activity might escape measurement, and the differences in loss of moisture between these and the periods of sleep might in reality be even larger than those actually recorded.

A calculation (not shown) of the heat loss in vaporization during the day (from 6 a. m. to 6 p. m.) and at night (from 6 p. m. to 6 a. m.) again suggests that the variations observed in the respective periods are related more closely to the activity of the infants than to extraneous factors of environmental temperature and humidity. The average proportion of heat lost in vaporization was lower at night in six of the eight observations and averaged 30.2 per cent for all observations as against 32.8 per cent for the daytime periods. The infants were awake during 17 and 35 per cent of the respective periods, the environmental humidities averaging 41 and 49 per cent and the temperatures 24.7 and 24.9 C., respectively. The relationship between the level of relative

humidity and the magnitude of the amount of water vaporized may, as mentioned before, again be fortuitous, the error being due to incomplete removal of the excess of water eliminated incidental to increased activity and attributable to an inadequate ventilating system of the infant's respiration chamber.

The importance of activity as a factor in influencing the proportion of heat lost by infants in the vaporization of water is reflected in the results obtained for the entire duration of calorimetric observations of from twenty and one-half to twenty-two hours. In these observations, in which the infants slept during the major portion of their residence in the respiration chamber (from 65 to 81 per cent of the time), their heat loss in vaporization constituted from 27 to 41 per cent of their total heat production, averaging 31.5 per cent for all observations. In only three of the eight observations was the upper normal basal limit of 30 per cent exceeded (table 9). It is probable, as will be shown later, that with greater activity and in the absence of the artificial restrictions necessarily imposed on infants in prolonged measurements of energy metabolism with the present forms of apparatus, an even higher fraction of the total heat production than the average figure of 31 per cent found in these observations might be dissipated through the agency of evaporation of water.

The Constancy of the Insensible Loss of Moisture From Day to Day.—It has been shown that during a prolonged sojourn in the respiration chamber estimations of the daily heat production of infants cannot be made with any regularity from their measured insensible perspiration, or from its component, water vapor. It now remains to answer the far more practical question whether such estimations are possible when infants are not so confined but are living, as our subjects did in the earlier days of the experimental periods, in a relatively normal environment. In table 11, the twenty-four hour insensible perspiration of the infants measured with the balance on the two to four days preceding calorimetric observations in each period is compared with the values obtained on the final days during their residence in the respiration chamber.

The total insensible loss of weight of the infants was consistently and notably higher on the days spent outside the respiration chamber than on the days of confinement within it. From 117 to 219 more grams of water per day was eliminated as insensible perspiration in the earlier days of the periods, these average increments representing increases of from 29 to 40 per cent above the levels measured on the final days. In none of the observations did the insensible loss of weight on the final day of the period attain the levels recorded for any of the earlier days.

There is every reason to believe that the differences in magnitude of the total insensible loss of weight measured under the two sets of experimental conditions (outside and inside the respiration chamber) were due primarily to alterations in the output of water vapor and not to any changed relationship between the amount of carbon dioxide exhaled and the amount of oxygen absorbed ($CO_2 - O_2$), since the diet and, presumably, the composition of the metabolic mixture (respiratory quotient) were maintained constant throughout experimental periods and through adequately long foreperiods. Furthermore, previously cited calorimetric studies [7] have shown that relatively minor fluctuations in the activity of infants comparable to those noted on the different days

TABLE 11.—*Insensible Perspiration Measured with the Balance on Individual Days of Periods*

Experiment	Insensible Perspiration per 24 Hours, Gm.							
	Days Outside Respiration Chamber					Day Inside Chamber	Deviation	
	1	2	3	4	Average		Gm.	Percentage
166*	497	586	617	608	577	358	219	—38
167†	...	...	...	...	...	341	...	....
169	583	497	556	...	545	386	159	—29
170	441	420	422	...	428	272	156	—36
171	336	286	268	...	297	180	117	—40
172	454	498	503	...	485	308	177	—36
173	424	390	446	...	420	293	127	—30
174	413	394	...	...	404	287	117	—29
Average	450	439	469	...	451	298	153	—34

* In this observation approximately one month intervened between measurements on the days preceding and those on the day of the calorimetric study.

† The insensible perspiration was not measured on the days preceding this calorimetric observation. The figure obtained for the day while inside the respiration chamber (341 Gm.), therefore, is not included in the average.

of these observations were without effect on the level of the respiratory quotient.

One is hard put to explain the large differences in output of moisture from the skin and lungs on the days spent outside and inside the respiration chamber. An attempt was made to control all the factors which are known to affect the insensible loss of water in order that the two series of measurements might be made under comparable conditions. The diet and clothes, selected to obviate excessive warmth or chilling, were maintained constant throughout periods. While absolute control of the activity of infants was obviously impossible, their good health and comfort and the artificial restriction of movements required for the collection of excreta combined to stabilize their behavior and to reduce spontaneous variations in activity on the individual days of the periods. In the absence of an air-conditioned room, which became available only after the completion of these studies, fluctuations in

environmental temperature and humidity were inevitable, but the daily variations recorded in the two series of measurements were not marked, as is shown in table 12.

The divergent results cannot be ascribed to environmental temperature, since average measurements recorded at four hour intervals with the same thermometer both while the infant was outside and while he was inside the respiration chamber were approximately the same. The extent to which the differences in output of moisture observed under the two sets of experimental conditions is directly attributable to variations in relative humidity is not ascertainable from the data. Although the relative humidity of the surrounding air on the earlier days of the

TABLE 12.—*Effect of Environmental Conditions and Activity of Infants on the Insensible Perspiration Measured with the Balance*

Experiment		Number of Days	Environment		Average Rectal Temperature F.	Percentage of Total Period		Average Amount of Insensible Perspiration, Gm. per 24 Hours
			Average Temperature, C.	Average Humidity, Percentage		Awake	Restless* and Crying	
166	Outside	4	26.3	40	99.9	45	17	577
	Inside	1	26.1	53	99.4	32	..	358
169	Outside	3	24.1	38	98.9	55	13	545
	Inside	1	24.0	59	98.0	43	24	386
170	Outside	3	23.5	26	99.0	46	16	428
	Inside	1	23.3	35	98.0	31	15	272
171	Outside	3	23.7	20	98.5	52	22	297
	Inside	1	24.0	41	97.5	40	6	180
172	Outside	3	23.3	33	99.1	51	20	485
	Inside	1	23.9	40	98.4	32	13	308
173	Outside	3	23.3	27	98.6	47	12	420
	Inside	1	23.9	38	98.4	42	6	293
174	Outside	2	23.0	30	101.2	52	10	404
	Inside	1	26.8	45	101.6	32	2	287

* The time occupied by eating is included in this column.

period was invariably lower than that recorded for the air within the respiration chamber on the final day—a condition which might have tended to cause a lower insensible output of moisture in the latter periods—the measurements were made with two different instruments (psychrometer and hair hygrometer, respectively) which, unfortunately, were not calibrated against each other. Calculations with the aid of standard tables [18] of the relative humidity of the air in the chamber from the moisture content of ten liter samples withdrawn at the start and at the end of each period of calorimetric measurement (residual tubes) revealed that the readings obtained with the hair hygrometer were always too high. The significance of the differences in the relative humidity recorded with the psychrometer and with the hygrometer

18. Marvin, C. F.: Psychrometric Tables for Obtaining the Vapor Pressure, Relative Humidity and Temperature of the Dew Point, U. S. Dept. Agric., Weather Bureau, 1915, table 12, p. 84.

on the days when the infant was outside and when he was inside the chamber, respectively, are, therefore, diminished. In favor of the view that the correlation between the level of relative humidity and the magnitude of the insensible loss of water may be fortuitous in nature is the previously cited work with infants [13] showing that fluctuations of relative humidity within the limits present in these observations both on the days when the infant was outside and on those when he was inside the respiration chamber were without significant effect on the insensible perspiration. The average rectal temperature, taken at four hour intervals, was higher in all except one observation (experiment 174) on the days preceding calorimetric measurements, but the differences, which in no instance exceeded 1 F., would scarcely seem to be of sufficient magnitude to explain even in slight measure the tremendously higher losses of moisture on these days.

Variations in the activity of the infants would again seem to offer the most tenable explanation for the large differences in the insensible loss of moisture on the days spent outside and inside the respiration chamber. As might have been expected, the infants were awake for longer periods of the day when they were free from confinement in the respiration chamber, but inspection of table 12 shows that in no instance were they restless or did they cry for even appreciable fractions of these days. The moderate degree of these increases in activity makes one hesitate to ascribe to this factor alone the enormous augmentations in loss of moisture from the skin and lungs measured on the days spent outside the respiration chamber, but no other explanation is available. The continuous flow of air to which the infants were exposed in the respiration chamber and not on the earlier days of the periods should theoretically have tended to increase the insensible output of moisture on the days spent inside the respiration chamber.

Irrespective of the reason, it is obvious that any attempt to estimate the twenty-four hour heat production of the infants while outside the respiration chamber according to the method suggested for adults under equivalent conditions would be doomed to failure.

The data collected in table 13 reveal the enormous error involved in such estimations. In this table, the average twenty-four hour heat production of the infants for the days spent outside the respiration chamber was computed from the calorimetric data in the manner previously described, allowance being made for the variations in activity on each of the days preceding the direct measurements. The average results thus obtained were compared with the average twenty-four hour values predicted from their insensible loss of water measured with the balance on these days. The discrepancies in the two series of figures ranged from 285 to 770 calories per day, or from 67 to 135 per cent.

Even when a hypothetic elevation in heat production of 100 per cent above sleeping levels was allowed in estimating the caloric expenditure of the infants for the portions of the earlier days of the periods while they were awake (analogous to the method described in an earlier section), the deviations between the average twenty-four hour heat production, calculated from the calorimetric data and predicted from the insensible loss of water, remained at the significantly high levels of from 21 to 95 per cent.

TABLE 13.—*Comparison of Heat Production for the Days Outside the Respiration Chamber Calculated from Calorimetric Data (Obtained on Final Day of Each Period) and That Calculated from the Insensible Perspiration Measured with the Balance (According to the Method Employed in Adults) in Terms of Averages per Twenty-Four Hours*

		Calculated from Calorimetric Data							Calculated from Insensible Loss				Deviation	
		Asleep			Awake									
			Calories			Calories								
Experiment	Number of Days	Hours	Per Hour*	Total	Hours	Per Hour*	Total	Total Calories	Insensible Loss, Gm.	Carbohydrate in Diet, Gm.	Nitrogen in Urine, Gm.	Total Calories	Calories	Percentage
166	4	13.0	19.8	257.4	11.0	28.5	313.5	571	577	72.2	4.0775	1,341	770	+135
169	3	10.8	20.5	221.4	13.2	28.1	370.9	592	545	77.8	4.4630	1,069	477	+ 81
170	3	13.0	17.2	223.6	11.0	24.3	267.3	491	428	59.1	3.5150	988	497	+101
171	3	11.5	14.4	165.6	12.5	19.8	247.5	413	297	29.6	2.3420	698	285	+ 69
172	3	11.8	19.1	225.4	12.2	25.7	313.5	539	485	75.1	4.8165	1,109	570	+106
173	3	12.75	18.4	233.7	11.25	25.4	287.0	521	420	58.9	3.7132	967	446	+ 86
174	2	11.4	20.9	238.3	12.6	25.1	316.3	555	404	58.9	4.3637	927	372	+ 67
Total								3,702	3,156	431.6	27.2909	7,099		

* The calories per hour while the infants were asleep and while they were awake represent the average figures obtained by direct measurement in the respiration chamber on the final days of periods.

The conclusion is inevitable that healthy infants living under conditions simulating their normal environment lose moisture from their skin and lungs, the heat equivalent of which (0.584 calories per gram of water) is greatly in excess of 24 per cent of their total heat production. That the major portion of the water eliminated through these channels in these observations was in the form of vapor rather than in the liquid state (and therefore unaccompanied by the dissipation of heat) is suggested by the fact that visible globules of sweat, although carefully looked for, were observed only for relatively short periods usually after meals. Further, the gain in weight of the clothes, measured apart from the weight of the infant, in no instance exceeded 3 Gm. per day throughout experimental periods.

If one accepts as valid estimations of the total heat production of the infants for the entire twenty-one days spent outside the respiration chamber from the calorimetric data determined on the seven days spent

inside, the heat equivalent of the water vapor contained in the measured insensible loss of moisture averaged 47 per cent of the heat produced so estimated and ranged between 38 and 56 per cent for individual observations of from two to four days. These values, derived from the calculation shown below, compare with a range of from 27 to 40 per cent and an average of 31.5 per cent for the eight days of occupancy of the respiration chamber (table 9).

Calculation of the Composition of the Total Metabolic Mixture for the Twenty-One Days Spent Outside the Respiration Chamber (from Data in Table 13)

Total insensible loss of weight	3,156 Gm.	(balance)
Total number of calories	3,702	(from calorimetric data)
Carbohydrate	431.6 Gm.	(diet)
Protein	170.6 Gm.	(27.2909×6.25)
Fat	132.6 Gm.	$\left(\frac{3{,}702 - (431.6 \times 4.1 + 170.6 \times 4.1)}{9.3}\right)$

Derivation of the Average Percentage of the Total Heat Produced Lost in the Vaporization of Water

$I\,L = I\,W + (CO_2 - O_2)$

$I\,W = I\,L - (CO_2 - O_2)$

$I\,L = 3{,}156$ Gm. and $(CO_2 - O_2) = (0.41\,C + 0.08\,P - 0.08\,F)$

$I\,W = 3{,}156 - (0.41 \times 431.6) + (0.08 \times 170.6) - (0.08 \times 132.6)$

$I\,W = 2{,}976$ Gm.

Number of calories lost in the vaporization of water $= (2{,}976 \times 0.584) = 1{,}738$

Percentage of calories lost in the vaporization of water $= \frac{1{,}738}{3{,}702} = 47$

Derivation of the Revised Equation for Estimating the Composition of the Daily Metabolic Mixture of Infants Under Maintenance Conditions

$I\,L = I\,W + (CO_2 - O_2)$

$I\,W = \frac{\text{Cal.} \times 0.47}{0.584} = 0.804$ calories

$\text{Cal.} = (4.1\,C + 4.1\,P + 9.3\,F)$

$(CO_2 - O_2) = (0.41\,C + 0.08\,P - 0.08\,F)$

$I\,L = 0.804\,(4.1\,C + 4.1\,P + 9.3\,F) + (0.41\,C + 0.08\,P - 0.08\,F)$

$I\,L = 3.71\,C + 3.38\,P + 7.40\,F$

$F = \frac{I\,L - (3.71\,C. + 3.38\,P)}{7.40}$

Substitution of the average figure, 47 per cent, in place of 24 per cent, which is customarily employed in calculating the amount of fat in the metabolic mixture of adults, results in the derivation of a revised equation which would seem to be better adapted for calculating the fat content of the daily metabolic mixture of infants ($F = \frac{I\,L - (3.71\,C + 3.38\,P)}{7.40}$). Application of this revised equation to the experimental data reveals that the average twenty-four hour heat pro-

duction of the individual infants could be predicted from their measured insensible loss of weight with a markedly reduced error, ranging between — 18 and + 19 per cent (table 14), in lieu of the enormous errors of from + 67 to + 135 per cent shown in table 13.

It is highly improbable that the magnitude of error found in the results obtained in these observations with the use of the revised equation would be maintained within the same relatively narrow range if the equation were adopted as a routine method of prediction of the twenty-four hour heat production of infants living in their normal environment. The evidence which may be cited to support this belief is: (1) the daily variations in the magnitude of the insensible loss of moisture which were observed in the same infants under apparently

TABLE 14.—*Comparison of Heat Production for the Days Outside the Respiration Chamber Calculated from Calorimetric Data (Table 13) and from the Insensible Perspiration According to Revised Method in Terms of Averages per Twenty-Four Hours*

		Calculated from Calorimetric Data				Calculated from Insensible Loss by Revised Method					
		Metabolic Mixture				Metabolic Mixture				Deviation	
Experiment	Number of Days	Protein (Urine Nitrogen, × 6.25), Gm.	Carbohydrate in Diet, Gm.	Fat by Difference, Gm.	Total Calories	Protein (Urine Nitrogen, × 6.25), Gm.	Carbohydrate in Diet, Gm.	Fat $\left(\frac{L-(3.71\,C+3.38\,P)}{7.40}\right)$ Gm.	Total Calories	Calories	Percentage
166	4	25.5	72.2	18.3	571	25.5	72.2	30.1	680	109	+19
169	3	27.9	77.8	17.1	592	27.9	77.8	21.9	637	45	+ 8
170	3	22.0	59.1	17.0	491	22.0	59.1	18.1	501	10	+ 2
171	3	14.6	29.6	27.1	433	14.6	29.6	18.6	354	79	—18
172	3	30.1	75.1	11.6	539	30.1	75.1	14.1	562	23	+ 4
173	3	23.2	58.9	19.8	521	23.2	58.9	16.6	491	30	— 6
174	2	27.3	58.9	21.7	555	27.3	58.9	12.6	470	85	—15

strictly comparable conditions on the individual days preceding calorimetric observations (table 11), (2) the large differences in the insensible output of moisture which were measured on the days when the infants were outside and the days when they were inside the respiration chamber, with, so far as could be determined, relatively minor differences in the activity of the infants and with otherwise comparable conditions, (3) the likelihood that individual differences in the threshold of sweating might be encountered in different infants even with similar grades of activity and with exposure to the same dietary and environmental conditions and, finally, (4) the complete lack of information regarding the quantitative effect on both heat production and moisture elimination of the artificial restriction of movements required for the collection of the excreta in the form of urine and feces—an unknowable factor, which must always remain conjectural and which cannot because of its very nature be subjected to direct measurement.

COMMENT

Doubt has recently been cast on the validity of making predictions of the heat production of human subjects under conditions of maintenance and over long periods from their insensible loss of body weight measured with the balance—the method suggested by Newburgh and his colleagues for the estimation of the heat production of adults. According to its critics, the fallibility of the method lies essentially in the difficulty of controlling the multiplicity of factors capable of distorting the relationship existing between the two mechanisms when the subject is in the basal state.

It was believed that the comparative ease with which the extraneous factors constituting the infantile environment might be controlled during prolonged periods of measurement made the infant a particularly suitable subject for study. The relationship between heat production and heat loss in the vaporization of water from the skin and lungs found in these prolonged studies on infants under maintenance conditions may briefly be summarized as follows: 1. The proportion of heat lost by normal infants by this mechanism under the aforementioned conditions was in general higher than the average figure of 24 per cent accepted for adults and that of 27 per cent previously found for infants in the basal state. 2. The percentage of heat lost in this manner by the same infants on individual days and by different infants was inconstant under fixed conditions of diet and under relatively standard environmental conditions. 3. Within the range of conditions present in these observations, the observed variations were apparently dependent in large measure on the extent of muscular activity of the infants.

It is conceivable that under perfectly controlled and standard experimental conditions, a constant relationship may be shown to exist between the total amount of heat produced by infants over long periods and the fraction dissipated in the vaporization of water. That these ideal conditions were not achieved in these prolonged studies is evidenced by the variability in the relation between the two mechanisms found not only while the infants were outside but also while they were inside the respiration chamber, even under relatively standard experimental conditions. The difficulties encountered in attaining this theoretically ideal state militate against the practicability of routine application to infants of any method of prediction of heat production over long periods based on the vaporization of water from their skin and lungs.

It seems reasonable that the adoption of such methods for infants must await precise information regarding the quantitative effect of extraneous factors and especially of activity on the mechanism of vapor-

ization and on the threshold of sweating as well as on the physical processes of radiation and convection of heat in these subjects.

SUMMARY AND CONCLUSIONS

Analysis of the daily diet, urine and feces and measurements of the twenty-four hour insensible loss of body weight (insensible perspiration) of three normal infants were made in eight observations of from three to five days each, under constant conditions of diet. In addition, their twenty-four hour heat production was concurrently determined by indirect calorimetry on the final day of each experimental period. The composite data were suitable for appraising the applicability to infants of the method proposed for predicting the heat production of adults over long periods from their insensible perspiration measured with the balance. The experimental evidence also permitted a critical analysis of each of the assumptions involved in the calculations. The results may briefly be summarized as follows:

1. Under fixed conditions of diet, the excretion of nitrogen in the urine is an accurate measure of the protein catabolism of infants.

2. Under fixed dietary conditions, the amount of carbohydrate of the diet is a reliable index of the amount of carbohydrate in the metabolic mixture, proving that infants as well as adults are essentially carbohydrate burners.

3. Healthy infants dissipate a higher proportion of the heat produced in the vaporization of water from the skin and lungs in prolonged (as opposed to basal) calorimetric observations and also when living in their normal infantile environment than 24 per cent, which is generally accepted as the percentage so eliminated by adults under equivalent conditions. The average values as found in these observations were 31.5 and 47 per cent, respectively, under the two sets of experimental conditions.

4. The average percentages, as found, were not constant for individual infants nor were they consistent for the same infant on different days. Within the range of environmental temperature and humidity present in these observations, the observed fluctuations between the two mechanisms tended to parallel the activity of subjects, but the relationship was by no means a quantitative one.

The conclusion seems warranted that the method employed for the estimation of the heat production of adults over long periods from their measured insensible loss of weight is not applicable in its present form to infants.

525 East Sixty-Eighth Street.

GROWTH OF ORIENTAL CHILDREN IN SAN FRANCISCO

A CONTRAST

MARY I. PRESTON, M.D.
SAN FRANCISCO

INTRODUCTION

As is well known, studies of most races are showing increased weight and stature of children as compared with the older generations, not only after immigration to America but in their native countries as well. Higher standards of living and the newer knowledge of nutrition have been thought responsible for these changes.

Briefly, white schoolboys of the San Francisco bay region [1] have increased 2 inches (5.08 cm.) in average height and 2 pounds (0.9 Kg.) in average weight in thirty years,[2] while the girls have advanced 1¾ inches (4.5 cm.) in average stature and 3 pounds (1.3 Kg.) in average weight. The gain of Japanese children in California in twenty years [3] surpasses this, but the Chinese children of San Francisco prove to be the exception to the rule; not only has there been no change of statistical significance from the figures of sixteen years ago from China,[4] but after 9 years of age these children do not reach the present-day standards in Canton,[5] the province from which the Chinese of San Francisco come, and are strikingly outdistanced by the Cantonese Chinese in Hawaii.[6]

SOCIAL-ECONOMIC STATUS

Chinese.—The factors contributing to the present physical status of the Chinese children of San Francisco are noteworthy.

From the Department of Pediatrics, Stanford University School of Medicine.

1. Pryor, H. B., and Stolz, H. R.: Determining Appropriate Weight for Body Build, J. Pediat. **3**:608 (Oct.) 1933.

2. Boas, F.: Growth of Toronto Children, Rep. Brit. A. Adv. Sc. August, 1897, p. 443.

3. Iyenaga, T., and Sato, K.: Japan and the California Problem, New York, G. P. Putnam's Sons, 1921; quoted from Spier.

4. Whyte, G. D.: Height, Weight and Chest Measurements of Healthy Chinese, China M. J. **32**:210, 1918.

5. Appleton, V. P.: Further Study of the Growth of Chinese, China M. J. **40**:259, 1926.

6. Keys, N., and Cadbury, W. W.: Age-Height-Weight Study of Cantonese School boys, China M. J. **40**:14, 1926.

The area occupied by the 30,000 Chinese in San Francisco is definitely restricted to an old part of the city with dark, narrow streets and alleys. Part of this restriction is due to the natural conservatism of the Chinese people in clinging to their old customs and manner of living, as well as to their native clannishness. In the rebuilding of Chinatown after the fire and earthquake in 1906, tenement houses of several stories were put up, each floor consisting of long rows of rooms on either side of a hall. The majority of the families in Chinatown, which are large (having up to twelve members) live in one room only, in which the elders object to the one window being opened.

Shelves or bunks on the walls are utilized as sleeping spaces, without mattresses. There is a community kitchen on each floor or sometimes only one to a building. A common toilet on each floor serves both sexes. The care of the kitchens, toilets and halls is often no one's business, and, as such, their condition can best be left to the imagination. There is no heat in the houses, and there is not even hot running water. However, the Chinese are very clean about their persons. Washing is hung on the roofs between the rows of drying meat and also along the halls against the walls. Piece-work of the sweat-shop variety has been only partially done away with in the homes. Tuberculosis is common.

Chinese parents, like some of the Russians, Greeks and Italians in San Francisco, insist that all children from kindergarten age up, attend language school to study their native language. Sessions in Chinatown are held from 5 to 8 p. m. daily and from 9 a. m. to 1 p. m. on Saturday. This affects the sleeping and eating habits, besides cutting off time for play and being outdoors. The little children get to bed between 10 and 11 o'clock, the older ones, from 11 to 12 or later, and the parents, from 12 to 2. With the whole family crowded into one room, a child would have to be an unusual sleeper to profit by going to bed any earlier.

Breakfast is mostly an unknown quantity except for candy bars, as the majority of parents are sleeping when the American schools open, and the children get themselves up and off to school. The first meal is eaten at midday, the time being set by the school noon interval. This consists largely of polished rice with a small portion of Chinese vegetables and a little pork, varied once in a while with fish, sometimes with bean curds and, rarely, with salted eggs. The children drink, instead of tea, the water in which the rice is cooked. The next meal is after school, about 4 o'clock, and is similar in composition to the first; after this meal the children depart for language school. The third meal is a night snack. Little fruit is eaten, and almost no milk was used until it was furnished by the increasing federal relief of the last two years.

Outdoor play is conspicuous by its absence. There is little of the day left for it. There is only one playground, and that is a small one. Any yards that might be used for play are absent in this crowded district, and the streets are too narrow for more than traffic requirements. The playground of the American school in Chinatown allows a little freedom of movement, but, on the whole, one is reminded of sardine packing.

Prenatal care is a rarity. The food in pregnancy leaves much to be desired. Midwives are luxuries. Fathers often deliver the babies; sometimes the older girls do. One mother, according to the parish priest, was delivered by a girl of only 14.

The younger generation are making strenuous efforts to better conditions, but they are working against great odds and deserve commendation for what they have succeeded in doing. White persons will not rent to Chinese either in or out of Chinatown without demanding exorbitant rents, and in the present depression the results are pernicious, to say the least. Chinese quarters are supposed not to need the janitorial service, repairs or improvements that almost any dwelling for white persons would get. In short, the native submissiveness of the Chinese race has allowed great imposition and exploitation by the unscrupulous and neglect by the authorities (every one realizes the great amount of protestation needed to bring about changes anywhere), though the Chinese are naturally a clean, self-respecting people with a high regard for family integrity and standards.

Japanese.—The factors contributing to the present physical status of the Japanese children of San Francisco are of interest also.

The Japanese adopted western dress at once on entering America and took on such American characteristics as appealed to the different classes. They have insisted on living in houses originally built for white persons and located in no longer fashionable residential areas, which they have steadily penetrated throughout the city. This has prevented overcrowding and has given them well lighted, airy homes, for the most part, with more sunshine, owing to the wide streets and the architecture of the houses, than is available in the tenement houses.

Language school takes up directly after American school and, while it interferes with the amount of outdoor life, is over, at the latest, by 6 o'clock. The small children attend for shorter periods. This allows ample sleeping hours compatible with the growth demands of developing children.

Breakfast is served before school, all members of the family being up. Lunch and dinner follow, as is the American custom. This is an improvement on the irregularity of meals in Japan. The Japanese in San Francisco pride themselves on serving at least one American meal

a day, sometimes two. Polished rice with a small portion of vegetables and meat comprises most of the diet. Japanese vegetables are now grown in California, and some Occidental varieties are being adopted. Fruit is becoming more and more a part of the daily food, and a taste for milk is slowly being acquired. The effects of changes of diet are shown in the lessened number of strikingly deformed chests found in the young children as compared with the older ones. Legs are less bowed than in Japan, a matter of satisfaction to the parents, though in Japan also there is improvement. Of course abandonment of the old country practice of sitting on the floor during the long school hours has helped. The habit of eating sweets, candy bars especially, is entirely too prevalent for maintenance of the best standards of growth. Appetites become perverted, and teeth are very bad.

The diet in pregnancy is a weak spot in the improvement of the race. Practically every mother of the children included in this study was born in Japan, and the mother is the most difficult member of a family to induce to adopt strange items of food. The lack of a balanced diet is shown most conclusively in the poor bone and tooth formation of the children.

Should the Japanese continue to drink more milk, include more fruit and vegetables in the diet, correct deficiencies in the diet in pregnancy and eliminate enervating layers of clothing while in the heated American schoolrooms, improvement in development and growth should continue.

SOURCES AND NUMBERS

Care must be taken, if comparisons are to be made with studies conducted in China or Japan, that descendants of peoples from specified areas in these countries are considered. When the Japanese race migrated from central Asia to the islands that they now inhabit they found and mixed with the natives, who were far from homogeneous, varying from a tribe of short people on the southern islands to the tall people in the north, who are described as being more like Russians and as having color in their cheeks, such as is at times found in their descendants. The Japanese of San Francisco come from the province of Hiroshima, for the most part, while some come from Oyahama; both these provinces are in the south-central part of Japan. Figures given by the Japanese Department of Education for the whole of Japan are not to be compared with figures for the Japanese on the Pacific Coast. Neither are Japanese studies comparable which exclude the poorest classes, such as that made by Mishima and quoted by Misawa,[7] for, interestingly, Japanese outcasts have been much more successful in

7. Misawa, T.: A Few Statistical Facts from Japan, Pedagogical Seminary **16**:104, 1909.

America than Japanese of the better classes, according to the Japanese Association of San Francisco, avowedly on account of their greater aggressiveness.

In the winter of 1931, through the courtesy of the principals of a number of language and church schools, measurements were made of 740 Japanese children of San Francisco, 346 boys and 394 girls. All but 15 of these were born in the United States, although the parents of all but 6 were born in Japan, most of them in the province of Hiroshima and some in Oyahama. Chinese children to the number of 391—188 boys and 203 girls—were also measured. These were of Cantonese extraction, i. e., from southern China.

Children of all social and economic classes attend the schools in which these studies were made, making the number in the series a representative sampling of the Oriental population. Mimeographed questionnaires were sent home to be filled out as to birth dates and birthplaces, as well as to the home diet.

The children well enough to attend school were included in the study, but the records of those obviously in poor health and of those with noticeable endocrine disorders (not more than 3 per cent in all) were discarded.

The Japanese will be discussed first throughout this paper, since data concerning larger numbers have been secured; then the Chinese will be considered. Similarly, the girls will in general be discussed before the boys, for the same reason (the greater reliability of the observations), though the Chinese boys will be considered in comparing other series in which girls have not been measured.

METHODS

Measurements.—All the measurements were made by me, with the exception of a small number that were made by a teacher and a nurse.

The instruments used were Fairbank's scales transported from place to place, Hrdlička's large sliding metal calipers, a steel metric tape and Franzen's special calipers for measuring subcutaneous tissue.

Two practices require special attention in studying growth in Oriental children. The first is the method of calculating ages. An Oriental baby is counted a year old when born, and another year is added every January; this leads to such discrepancies as that of considering the age of the baby born on December 31 as 1 year at that time and as 2 years the following day, January 1, while by western standards the age would be recorded as 2 days only.

The other practice, due to the lack of heat in houses of the Orient, is the addition of layer after layer of clothing as winter approaches, until not uncommonly from three to seven layers, not counting outdoor wraps, must be peeled off before the single layer accepted for this study is reached. The majority of the children under 12 years of age were found to wear three woolen sweaters, at least, between the undershirt and the outer shirt or dress. Canton flannel garments usually form the other layers. That the children do not look greatly

overpadded is due to the fulness of their cheeks, often a deceptive index of the state of nutrition. As the children wear all this clothing in the heated, if not overheated, American schoolrooms, this practice presumably accounts to no small degree for the great prevalence of common colds. The older children, especially the Japanese, insist on wearing as little underclothing as their white neighbors, have fewer colds and are in much better physical condition than the Chinese. Clearly the prevailing practice in most studies on school children of weighing "with indoor clothing" would have led with children of the Far East to even greater inaccuracy, not to say material error, than has been pointed out for white children. Age was recorded from the nearest birthday; for example, all children between 8½ and 9½ years of age were considered to be 9 years old.

Clothing was reduced to one layer of underwear, usually cotton or rayon, which was found to weigh from 0.18 to 0.36 Kg. (0.4 to 0.8 pounds). This fraction was not subtracted in compiling the tables for weight, but as it was small the weight may be considered practically net. The stature was measured net. The underclothing was sufficiently adjustable to permit the measurements of the chest, bicristal diameter and fat to be made directly on the surface of the skin. In measuring the bicristal diameter the calipers were applied with firm pressure. The technic of Franzen [8] was followed in measuring the folds of fat, that of the arm being measured at the level of the short head of the biceps. In measuring the abdominal subcutaneous tissue the calipers were applied directly to the fold of fat extending from side to side just below the umbilicus, which was picked up to the right of the midline and one third of the distance from the umbilicus to the anterior superior spine of the ilium. This fat fold has been found to be consistently greater than that directly to one side of the umbilicus but bears an almost constant relation to the latter.[9] In a number of subjects heavy scars from appendectomies or extensive burns on the body interfered, in which children the measurements had to be taken to the left of the midline.

Weight was recorded to the nearest tenth of a pound and height to the nearest tenth of an inch; these measurements were later converted to the metric equivalents.

Deformities of the bones of the chest and limbs were recorded as +, ++ and +++.

The number of carious teeth was also counted for each child.

Statistical Methods.—In calculations the tables of Pearson [10] and of Barlow [11] and the Marchant calculating machine were used.

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8. Franzen, F.: Physical Measures of Growth and Nutrition, School Health Research Monographs, no. 2, New York, American Child Health Association, 1929.

9. It will be noted that, while Franzen's [8] method of measuring fat folds was followed, the site of the abdominal fat fold differs from that used by some other observers. An important aspect of measurements is comparison. The fat of 25 children of various ages was measured not only below the navel, which is termed here the standard method, but also at a point directly to the right of the navel. On plotting the latter measurements against the standard measurements, the points fit satisfactorily a straight line, which is represented by the equation $x = -1.35 + 1.29\ y$, in which x gives the probable value which would have been attained by the standard method, judging by the original observer's value y.

10. Pearson, Karl: Tables for Statisticians and Biometricians, London, Cambridge University Press, 1924.

11. Barlow, P.: Barlow's Tables, revised by L. J. Comrie, New York, Spon & Chamberlain, 1930.

Tables of constants were made out for the Japanese for height, weight, bicristal diameter and fat folds at each age from 5 to 15 years but not for the smaller number of Chinese, averages being used in their case. These tables contain: (1) the number of observations made at each age, as this is useful in evaluating the reliability of the results (the smaller the number the less reliable the result); (2) the arithmetical mean, as it gives the average for the group; (3) the standard deviation, which denotes how much the group varies around the average, expressed in absolute units such as centimeters; (4) the coefficient of variation, which gives that variation in percentage of the average, and (5) the probable error of the means, to show the random sampling of the population studied and therefore to indicate the reliability of the averages.

Most of the calculations were verified by being checked by an assistant.

RESULTS

In considering the charts, there are three factors besides sex and age that need to be considered, namely, race, region and period.

Stature.—A racial difference was noted in that the stature of the Japanese girls of San Francisco had not reached the stature of the white girls of the same region as determined in 1923 (Faber [12]) and had not reached the stature of the white girls of the same region and period by from 7 to 11 cm. (Pryor and Stolz,[1] including many figures from private medical practice), but it had reached and slightly exceeded, as a whole, the stature of the Chinese girls of San Francisco (chart 1; the measurements for the Chinese are not shown).

TABLE 1.—*Table of Averages of Measurements of Chinese Children*

	Girls				Boys			
Age, Years	Number	Height, Cm.	Weight, Kg.	Bicristal Diameter, Cm.	Number	Height, Cm.	Weight, Kg.	Bicristal Diameter, Cm.
4	10	97.5	14.7	16.3	8	100.8	16.1	17.0
5	14	103.3	16.2	17.4	27	104.8	17.5	17.8
6	23	107.3	17.7	17.6	19	107.7	17.7	17.9
7	16	115.7	19.8	18.8	8	111.8	19.1	18.3
8	15	118.5	21.0	19.4	13	120.8	22.0	19.3
9	34	122.4	23.1	20.0	20	126.0	23.1	19.8
10	34	127.3	24.8	20.5	38	127.1	25.2	20.0
11	22	131.1	28.0	21.5	27	132.2	27.5	21.3
12	20	138.6	31.5	22.7	10	134.6	29.5	21.7
13	5	144.5	35.3	24.8	7	139.7	32.4	22.2
14	3	145.8	38.4	25.6	7	146.7	35.6	23.0

A regional difference appears when the Los Angeles and San Francisco girls are compared, since the Japanese girls of the southern city were taller by about 1 cm. until the age of 13 (Suski,[13] in 1932). On

12. Faber, H. K., in Terman, L. M.: Genetic Studies of Genius, Stanford University, Calif., Stanford University Press, 1925, vol. 1, p. 45.

13. Suski, P. M.: Body Build of American Born Japanese Children, Biometrika **25**:322, 1933.

the other hand, the Los Angeles girls had no advantage in stature over the Japanese girls of a northern region, that of Seattle (Spier,[14] in 1923); these measurements are omitted for clarity from the charts, as those of the children from Los Angeles and those of the children from Seattle are practically the same.

The element of time is also involved, as is evident when one considers the advance shown by the Japanese girls in San Francisco when compared with the Japanese girls in California twenty years ago who,

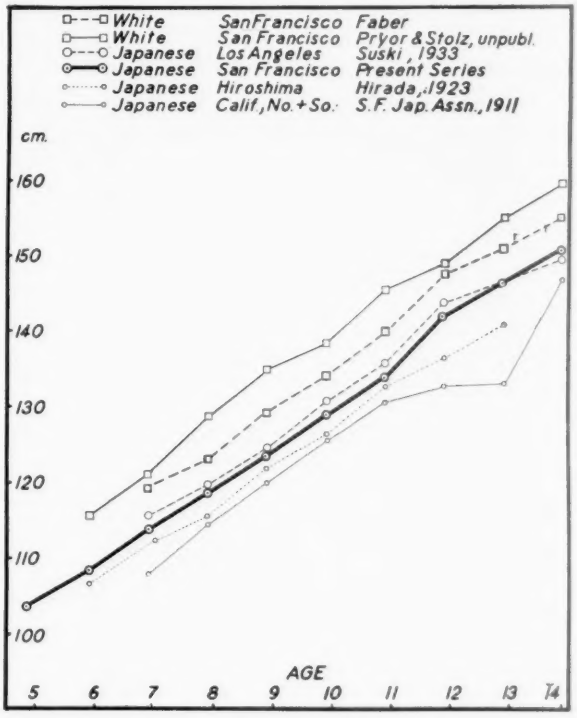

Chart 1.—Average statures of Japanese girls of different regions, compared with those of white girls in San Francisco.

in turn, it was noted, had shown gains over the children of the land from which they came (Iyenago and Sato,[3] in 1911). Both groups originally came from Hiroshima, the province from which the great majority of the Japanese on the Pacific Coast emigrated and in which a gain, though much smaller, has since taken place. Though the measurements of the Los Angeles children in 1932 equaled those recorded in Seattle in 1923, the honors are on the Seattle side, as the effect of the time

14. Spier, L.: Growth of Japanese Children Born in America and in Japan, Univ. Wash. Publ., Anthrop. **3**:1, 1929.

element of nine years cannot be disregarded. The same is true of the difference between the measurements of the two series of white children, taken ten years apart.

When Spier made his measurements in Seattle, he delegated a Japanese university student to make simultaneous ones in Hiroshima, thus eliminating the element of time, and found the stature of the Japanese children in Seattle to be superior; this was apparently a regional effect. Nothing has been found to explain the difference between the Los Angeles and the Seattle group.

TABLE 2.—*Constants: Weight and Stature of Japanese Children*

	Weight, Kg. (with 0.18 to 0.36 Kg. of Underwear)						Stature, Cm. (Net)					
Age, Years	Number	Mean	± Probable Error	Standard Deviation	± Probable Error	Coefficient of Variation	Number	Mean	± Probable Error	Standard Deviation	± Probable Error	Coefficient of Variation
Girls												
5	32	16.78	0.20	1.71	0.14	10.3	32	103.4	0.63	5.23	0.43	5.06
6	37	18.32	0.16	1.48	0.12	8.14	37	108.2	0.46	4.21	0.33	3.90
7	49	20.35	0.23	2.41	0.16	11.68	49	113.8	0.48	5.05	0.35	4.45
8	51	22.58	0.25	2.66	0.18	11.84	51	118.6	0.58	4.87	0.41	4.11
9	32	24.32	0.36	3.07	0.25	12.38	32	123.3	0.56	4.67	0.41	3.79
10	57	27.14	0.39	4.42	0.28	16.35	57	128.7	0.63	7.01	0.43	5.44
11	43	30.46	0.48	4.72	0.34	15.53	45	133.6	0.71	6.75	0.48	5.06
12	24	35.38	0.93	6.77	0.66	19.18	25	141.9	1.04	7.75	0.73	5.45
13	24	38.69	0.66	4.78	0.46	12.40	24	145.9	0.84	6.12	0.61	4.20
14	20	43.25	0.64	4.26	0.45	9.85	21	150.4	0.45	3.02	0.30	2.01
Boys												
5	30	17.66	0.56	2.06	0.17	13.59	31	105.2	0.58	4.85	0.41	4.60
6	30	18.98	0.30	2.44	0.21	12.90	30	108.9	0.61	4.97	0.43	4.56
7	32	21.54	0.23	1.92	0.16	8.95	32	115.3	0.56	4.67	0.41	4.05
8	45	22.52	0.20	2.00	0.14	8.89	45	118.8	0.38	3.78	0.27	3.19
9	48	24.01	0.24	2.48	0.17	10.38	48	123.1	0.58	5.94	0.41	4.83
10	33	26.70	0.40	3.44	0.28	12.90	35	127.7	0.81	7.03	0.56	5.52
11	30	28.78	0.48	3.89	0.34	13.58	30	132.8	0.76	6.09	0.53	4.60
12	26	34.68	0.73	5.52	0.52	15.95	26	140.7	0.99	7.08	0.66	5.03
13	22	36.23	0.95	6.61	0.67	18.29	22	142.2	0.96	6.68	0.68	4.69
14	11	44.83	2.02	9.95	1.43	22.23	11	152.1	1.77	8.76	1.27	5.75

The white and the Japanese girls both showed a vigor of prepubertal growth that was lacking in the Chinese. This up-push occurred in the white girls at 11 years, a year earlier than in the Japanese.

The same relative positions on the charts are held by the boys as by the girls (chart 2), with the Los Angeles boys a little taller and the Chinese boys of San Francisco showing a progressive slowing after the eleventh year (chart 3). A prepubertal spurt was manifested only by the Japanese boys of San Francisco.

Other studies of the stature of Chinese boys were available for comparison, including two made in Canton, China, the province from which the Chinese of the Pacific Coast and Hawaii emigrated, one by

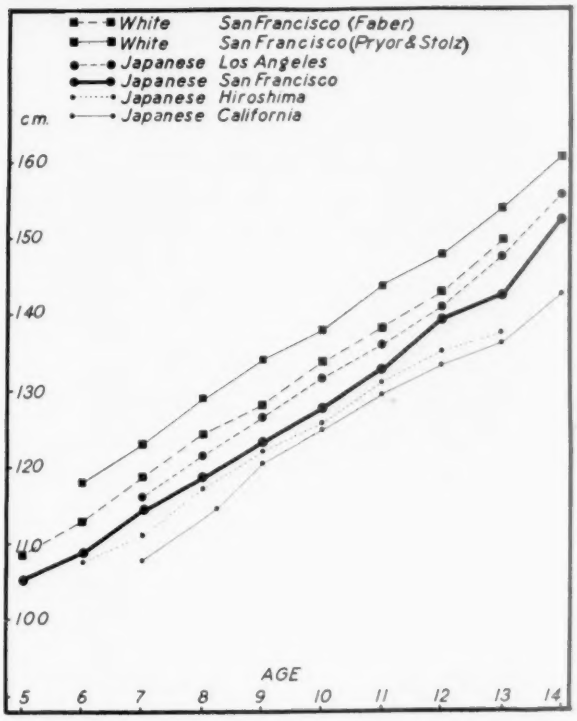

Chart 2.—Average statures of Japanese boys of different regions, compared with those of white boys in San Francisco.

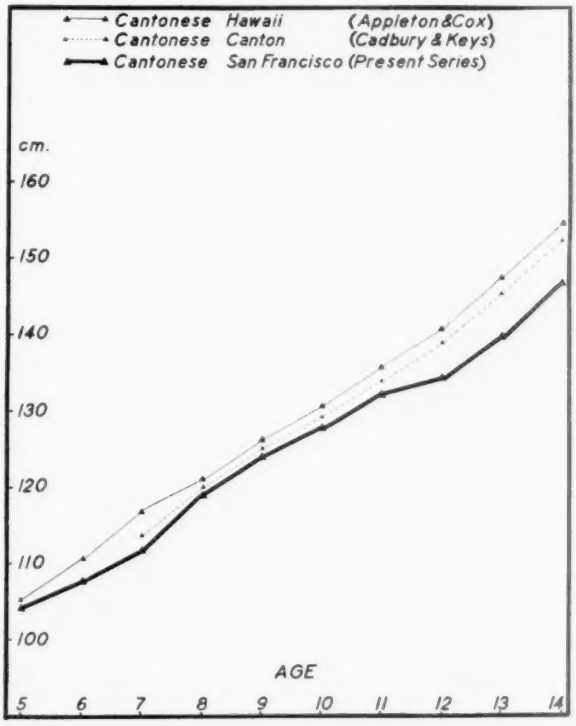

Chart 3.—Average statures of Chinese boys of Cantonese ancestry living in different regions.

Keys and Cadbury [6] and one by Stevenson [15] (the measurements found in these studies are not charted here, as the differences between the two are not of statistical significance). The Chinese in Hawaii were the tallest, and the Chinese in California, the shortest. This is surprising, as the latter might be expected to be larger than the Cantonese in China, but those included in this study, at least, fell behind at each age, especially in adolescence (8 cm.).

Weight.—The weight of the Japanese children of San Francisco (girls, chart 4; boys, chart 5) was not as great as that of the white

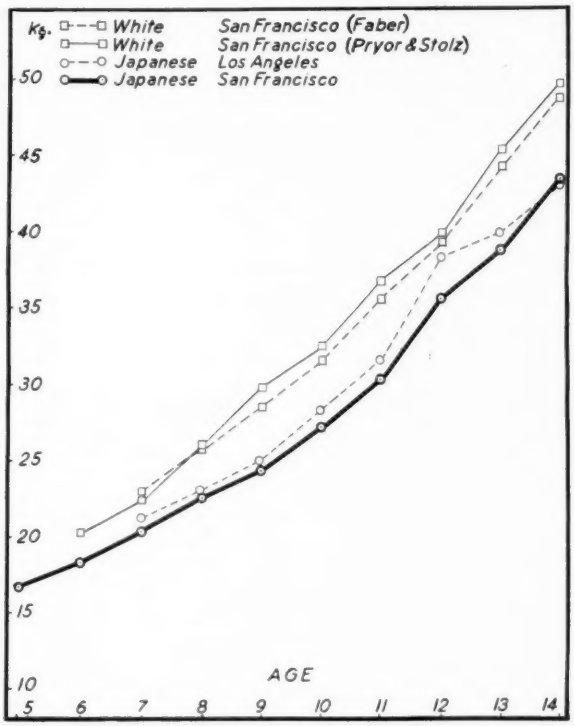

Chart 4.—Average weights of Japanese girls of different regions, compared with those of white girls in San Francisco.

children of the same region and was slightly less than that of the Los Angeles children but was considerably above that of the Chinese (chart 6). A prepubertal spurt at 12 years was noted in the Japanese of both sexes and in the Japanese girls in Los Angeles, and one was noted in the white girls at 13 years. A definite acceleration took place during the fourteenth year in both white and Japanese children but not in the Chinese children of either sex.

15. Stevenson, P. H.: Collected Anthropometric Data on the Chinese, China M. J. **39**:855, 1925.

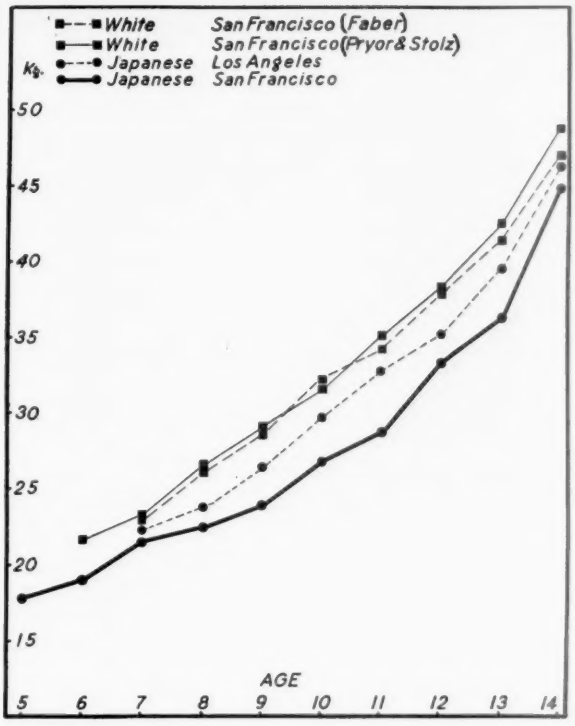

Chart 5.—Average weights of Japanese boys of different regions, compared with those of white boys in San Francisco.

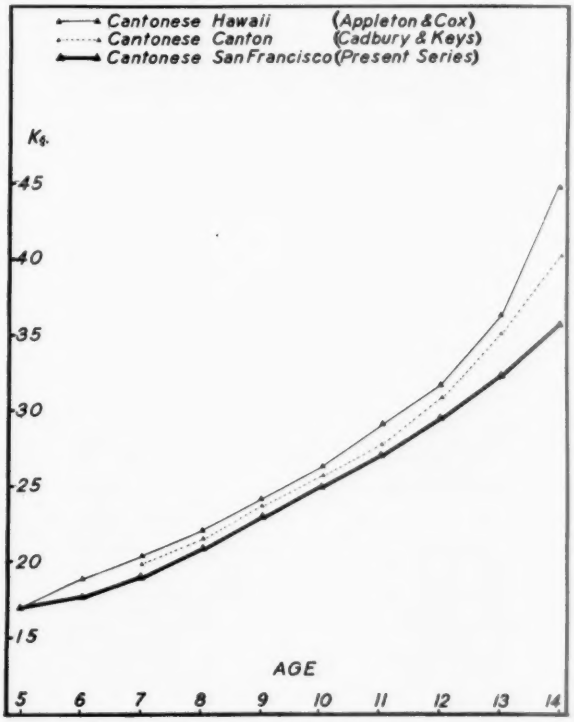

Chart 6.—Average weights of Chinese boys of Cantonese ancestry living in different regions.

The Cantonese of Hawaii, China and San Francisco showed parallel curves for gains in weight, those of Hawaii being heaviest, those of China less heavy and those of San Francisco least heavy. Again it will be observed that the acceleration period from 13 to 14 years, found in the Chinese of both Canton and Hawaii, was lacking in those of San Francisco. The greater abundance and variety of food, especially fruits, combined with an immeasurably greater amount of sunshine, probably accounts for the superiority of the Chinese children in Hawaii

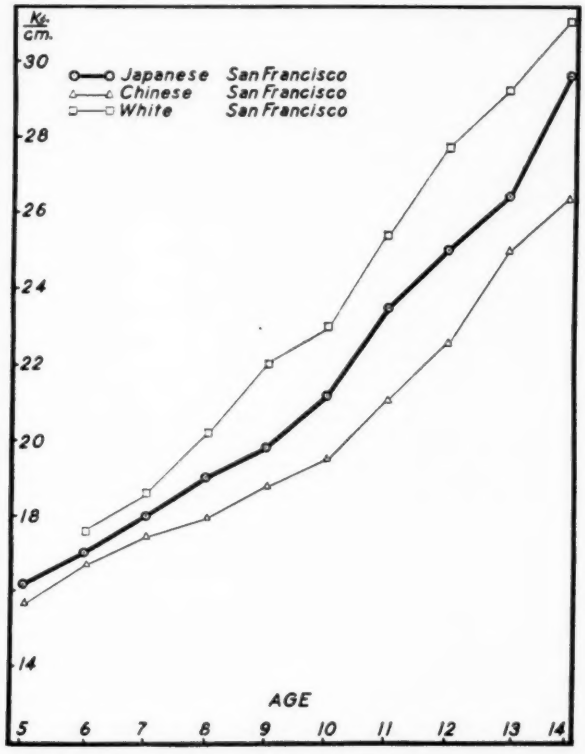

Chart 7.—Ratios of weight to stature of Japanese, Chinese and white girls living in San Francisco.

over the children of Chinatown in San Francisco, with its health violations of dark, cold, overcrowded dwellings, inadequate food ill-suited to growing children's needs, brief sleeping hours and little sunshine or play time.

Ratio of Weight to Stature.—For both boys and girls, the three curves maintain the same relative positions as in the curves for both weight and stature, white children showing the highest ratio, the Japanese children showing the next highest ratio and the Chinese children lagging behind (charts 7 and 8).

Bicristal Diameter.—Except for an interval from 8 to 11 years, the Japanese girls in San Francisco had a greater pelvic width than the white girls, and this was true to an increasing degree after 11 years of age.

Parallel to the lack of any periods of increased vigor of growth in weight and height shown on the part of the handicapped Chinese, the same situation holds with the boys in regard to the bicristal diameter, though the white and Japanese boys both showed spurts of growth in

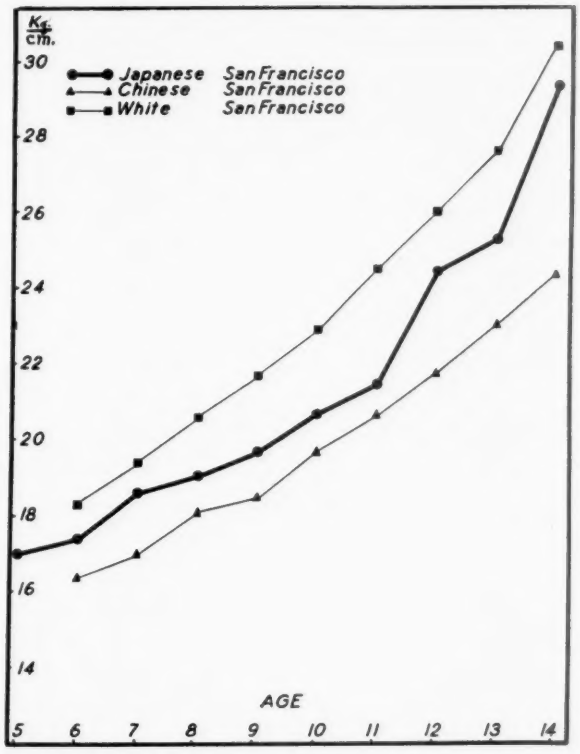

Chart 8.—Ratios of weight to stature of Japanese, Chinese and white boys living in San Francisco.

pelvic width. However, with the girls, strong biologic forces appear to have been centered in bringing about an increased pelvic width in the thirteenth year, at a distinctly accelerated rate (chart 9).

The pelvic width of the white boys was greater than that of any others; the Japanese boys of San Francisco showed the next greatest measurements, and the Japanese boys of Los Angeles dropped to third place (chart 10).

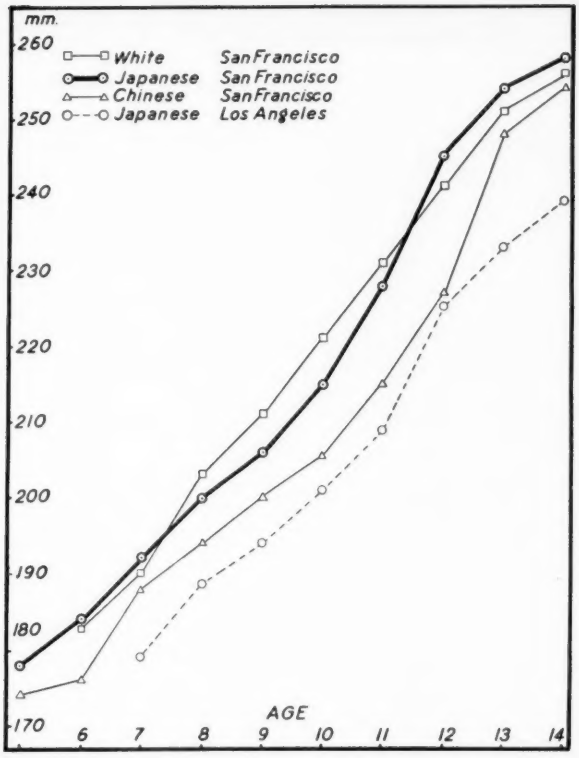

Chart 9.—Bicristal diameters of Japanese, Chinese and white girls of different regions.

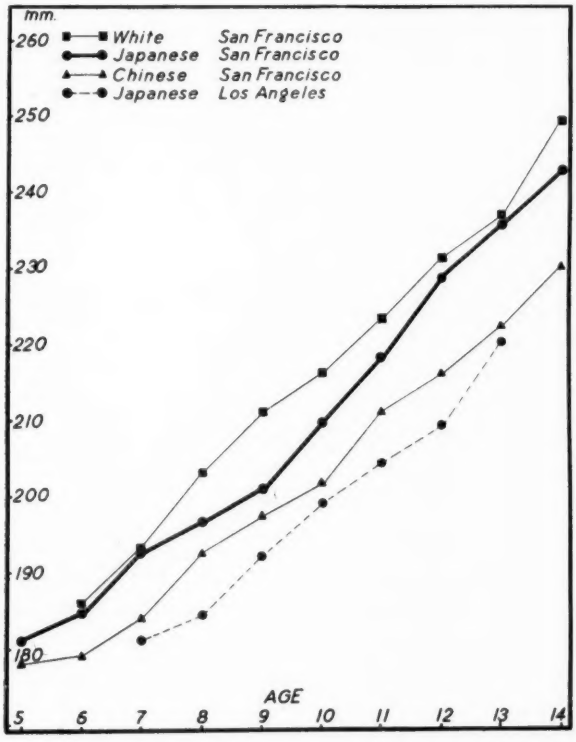

Chart 10.—Bicristal diameters of Japanese, Chinese and white boys of different regions.

To obtain a better picture of racial and sex differences in bicristal development, five curves, including one from Gray and Ayres'[16] study of private school children, have been depicted in chart 11. The boys had an early lead in pelvic width, which they lost at different ages in each race; the pelvic width of Japanese boys and that of Japanese girls of both Los Angeles and San Francisco coincided at the age of 7 years. A period followed in which the two sexes were of the same pelvic width for an interval up to two years, and then the girls began to show a notable widening. The sex difference during adolescent development varied with each race and can best be compared by inspection of the shapes of the curves. A pronounced difference was seen between the Japanese of Los Angeles and those of San Francisco. The Japanese girls of both sections showed an initial spurt in the twelfth year, but the period of sex difference was of shorter duration in the girls of the

TABLE 3.—*Constants: Bicristal Diameter of Japanese Children*

	Girls						Boys					
Age, Years	Number	Mean	± Probable Error	Standard Deviation	± Probable Error	Coefficient of Variation	Number	Mean	± Probable Error	Standard Deviation	± Probable Error	Coefficient of Variation
5	32	17.79	0.11	0.88	0.07	4.93	31	18.10	0.14	1.18	0.10	6.52
6	37	18.42	0.09	0.78	0.06	4.24	30	18.47	0.10	0.82	0.07	4.44
7	49	19.25	0.11	1.17	0.08	6.09	32	19.27	0.12	1.03	0.08	5.32
8	51	20.07	0.10	1.03	0.07	5.15	45	19.61	0.09	0.93	0.07	4.75
9	32	20.58	0.12	1.04	0.09	5.03	48	20.08	0.11	1.09	0.08	5.44
10	57	21.53	0.13	1.46	0.09	6.76	33	20.98	0.14	1.16	0.09	5.55
11	45	22.81	0.15	1.52	0.11	6.66	30	21.80	0.16	1.27	0.11	5.82
12	25	24.51	0.26	1.90	0.18	7.77	26	22.82	0.27	1.22	0.11	5.36
13	24	25.4	0.19	1.43	0.14	5.64	22	23.58	0.21	1.45	0.15	6.15
14	21	26.46	0.15	1.05	0.11	3.98	11	24.21	0.55	2.74	0.39	11.43

South (climate?), and in the boys also, since the boys had the same bicristal diameter as the girls at 14, though they did not continue to forge ahead to the greater adult width, as did the white boys in private schools, who passed the girls at 16. Whether a southern climate tends to shorten the period of pubertal change and affect the pelvic width, as is suggested by Suski's figures, the Japanese of both areas being of the same stock, needs further investigation.

Bicristal Diameter Relative to Stature (Width-Length Ratio).—To allow for stature it is often useful to examine "relative" measurements, in this case the ratio of the bicristal diameter to the stature. The value was quite different in the three races (chart 11). The Japanese had the highest ratio; the Chinese took an intermediate position, and the white children had the lowest ratio, as their stature was the greatest.

The age changes are noteworthy. The two sexes behaved alike up to the age of 10 years, with the width-length ratio, Bc/S, falling as the

16. Gray, H., and Ayres, J. G.: Growth in Private School Children, Behavior Research Fund Monograph, Chicago, University of Chicago Press, 1931.

stature increased faster than the bicristal diameter, until the age of 7 to 8 years, and then remaining fairly stationary until about the age of 11. Thereafter, the curves for the boys continued slightly downward, but those for the girls, who showed an increasing pelvic width, returned nearly to the high ratio of early life, giving the graphic curve a bowl-like contour.

As can be seen readily by inspection of chart 12, there was so pronounced a difference between the white children and the Japanese chil-

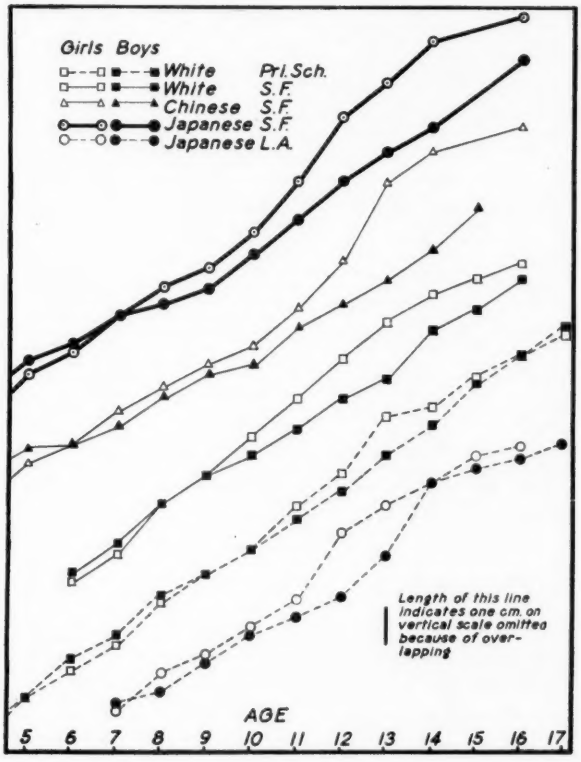
Girls Boys
White Pri.Sch.
White S.F.
Chinese S.F.
Japanese S.F.
Japanese L.A.
Length of this line indicates one cm. on vertical scale omitted because of overlapping
AGE
5 6 7 8 9 10 11 12 13 14 15 16 17

Chart 11.—Racial and sex differences in bicristal diameter among Japanese, Chinese and white children in different regions. Pri. Sch. indicates private schools; S. F., San Francisco, and L. A., Los Angeles.

dren in this ratio of bicristal diameter to stature that the value of the tables of Pryor and Stolz showing the ratio of the width to the weight (based on measurements of white children) is considered of unquestionable value for predicting the weight of the Japanese (chart 13*A*, in which the figures of Lucas and Pryor [17] for the ratio of width to

17. Lucas, W. P., and Pryor, H. B.: Physical Measurements and Physiological Processes in Young Children: Some Correlations, J. A. M. A. **97**:1127 (Oct. 17) 1931.

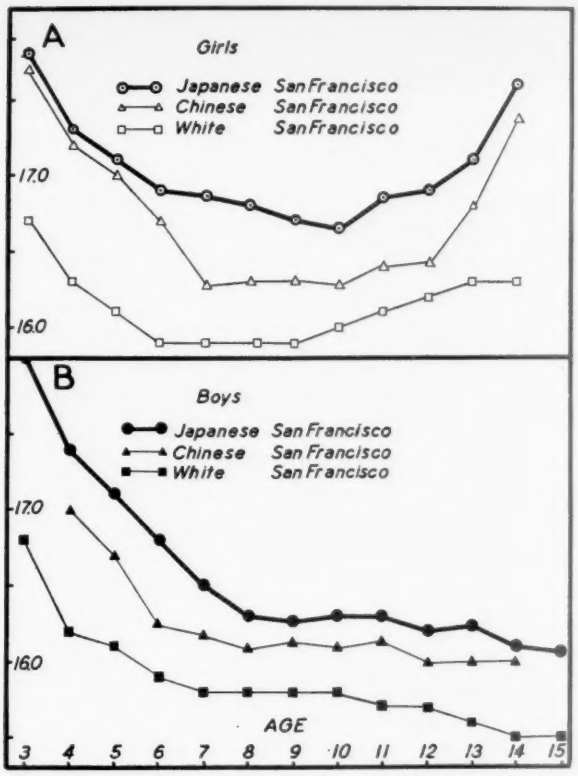

Chart 12.—Ratios of bicristal diameter to height in Japanese, Chinese and white children in San Francisco. *A* shows the ratios for girls, and *B* shows the ratios for boys.

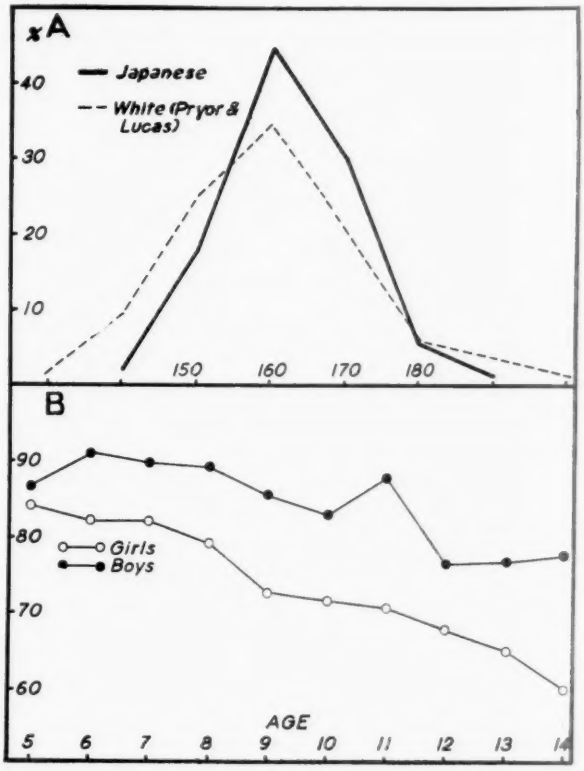

Chart 13.—*A*, ratios of the bicristal diameter to height in Japanese and white children. *B*, ratios of the thickness of the arm fat fold to that of the abdominal fat fold in Japanese boys and girls.

length, excluding values for babies, are used for comparison, since the numbers and the distribution according to age are similar to those of the present series). In this chart it will be seen that there is a decided shift of the curve for the Japanese children from that of the white children. The racial difference in the time of onset of the prepubertal acceleration in increase of pelvic width and also the suggested difference in the length of the pubertal changes due to climatic influence would, it seems, also interfere with the prediction value of these tables.

Fat Folds.—On the arm, the mean thickness of the fat fold picked up over the short head of the biceps showed a surprisingly slight increase

TABLE 4.—*Constants: Thickness of Fat Folds of Japanese Children*

		Arm Fat Folds					Abdominal Fat Folds				
Age, Years	Number	Mean	± Probable Error	Standard Deviation	± Probable Error	Coefficient of Variation	Mean	± Probable Error	Standard Deviation	± Probable Error	Coefficient of Variation
Girls											
5	32	30.91	0.50	4.22	0.36	13.65	36.97	0.64	5.36	0.45	14.50
6	37	31.36	0.48	4.31	0.34	13.72	38.48	0.71	6.43	0.50	16.72
7	49	31.56	0.39	4.05	0.28	12.82	38.56	0.73	7.56	0.52	19.59
8	51	31.75	0.39	4.11	0.27	12.94	40.25	0.73	7.72	0.52	19.18
9	32	32.40	0.59	4.99	0.42	15.39	44.87	1.42	11.91	1.00	26.54
10	57	33.85	0.44	4.88	0.31	14.41	47.43	1.07	12.02	0.76	25.35
11	45	36.64	0.61	6.03	0.43	16.48	52.08	1.26	12.57	0.89	24.15
12	25	34.96	0.87	6.48	0.62	18.53	51.64	2.05	15.16	1.45	29.37
13	24	33.88	0.47	3.41	0.33	10.07	52.13	2.17	15.98	1.55	30.65
14	21	36.07	0.58	3.95	0.41	10.94	60.60	2.14	14.51	1.51	23.95
Boys											
5	31	28.44	0.48	3.97	0.34	13.95	32.92	0.63	5.22	0.45	15.87
6	30	28.56	0.51	4.11	0.36	14.40	31.50	0.54	4.37	0.38	13.88
7	32	28.93	0.50	4.21	0.35	14.54	32.31	0.57	4.78	0.40	14.79
8	45	27.61	0.38	3.83	0.27	13.88	31.14	0.43	4.24	0.30	13.63
9	48	28.27	0.36	3.69	0.25	13.04	33.13	0.65	6.70	0.46	20.23
10	33	29.64	0.50	4.25	0.35	14.34	36.14	0.87	7.47	0.62	20.58
11	30	30.00	0.65	5.26	0.46	17.53	34.26	0.81	6.57	0.57	19.17
12	26	30.31	0.79	5.97	0.56	19.71	39.76	1.41	10.67	0.98	26.81
13	22	30.45	0.75	5.23	0.53	17.17	39.81	1.22	8.48	0.86	21.30
14	11	28.02	0.52	2.59	0.37	9.26	36.36	1.54	7.61	1.09	20.94

with age, from 32 mm. at the age of 5 years to 37 mm. at the age of 14 years in girls—an increase of only 5 mm.; and from 27 to 31 mm. in the boys at these ages—an increase of only 4 mm. Abdominal fat folds extending laterally below the umbilicus, on the other hand, showed an increase in thickness in the same age periods of 23 mm. in the girls and 7 mm. in the boys. The amount of fat on the arm reported by Franzen [8] for white boys and girls varied distinctly from that for the Japanese (chart 14); the white children apparently have much less subcutaneous fat than do the Japanese.

The Japanese girls showed a prepubertal rise in the arm fat between the ages of 10 and 11, i. e., a year earlier than their other periods of acceleration. Their remarkable prepubertal increase in the abdominal fat likewise took place by the eleventh year, while the increase for the

boys took place in the twelfth year. Changes in the fourteenth year further enlarged the subcutaneous layer of fat in the girls, and in the sixteenth year (the figures for this period are not shown) the thickness of the fat became still more pronounced in the arm fat and even more so in the abdominal fat, reaching a new high record of 67 mm. The thickness of the arm fat in the boys, which decreased after the thirteenth year, remained low, the play of underlying muscles showing clearly

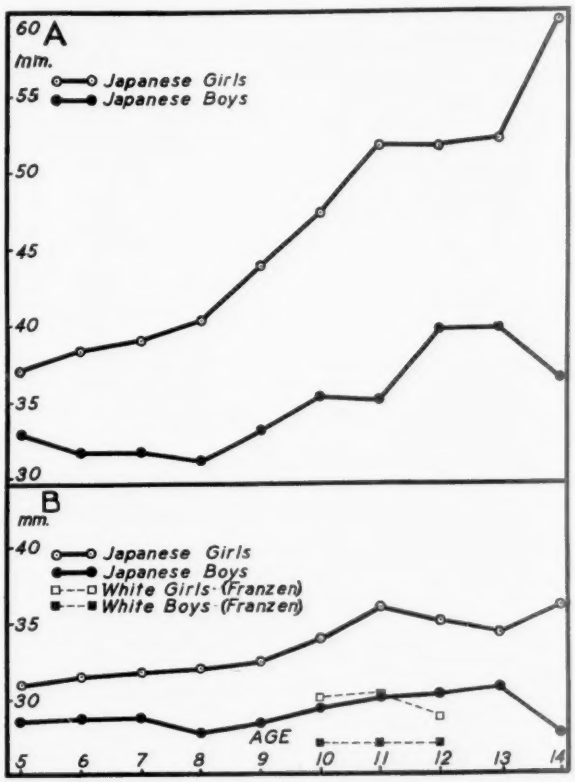

Chart 14.—*A*, thickness of the abdominal fat fold in Japanese boys and girls in San Francisco. *B*, thickness of the arm fat fold in Japanese children and white children.

beneath the thin skin; the surprise came in finding a generous roll of fat below the umbilicus, averaging 7 mm. thicker than that noted at 14 years of age. That the arm fat in boys tends to remain less after the thirteenth year was borne out in measurements, not published here, of 19 boys at the ages of 15, 16, 17, 18 and 19, giving the impression that the adult masculine contour of the arm is reached at that time, in contradistinction to the feminine well padded, smooth arm surfaces.

The ratio between the arm fat and the abdominal fat (chart 13*B*) changed from year to year in both the boys and the girls, indicating that the amount of arm fat cannot be taken as a criterion of the amount of other subcutaneous fat. In the girls the ratio varied from 90 to 76, and in one year, the twelfth, it dropped from 87 to 76. In the boys the ratio fell fairly steadily from 84 at 5 years of age to 60 at 14. Furthermore, if the arm fat were a reliable index of the amount of the subcutaneous body fat, the correlation with the fat of other regions should be very high and should be the same for all periods. The correlation between the arm fat and the abdominal fat was 0.62 ± 0.02 for 288 girls from 3 to 10 years of age and 0.75 ± 0.03 for 106 girls from 10 to 17. These correlations do not strike one as being high enough to warrant use of the arm fat in estimating the amount of other subcutaneous fat.

In 15 underweight white children who were measured more than once in the pediatric clinic the average gain was 4 mm. in the abdominal fat and 1 mm. in the arm fat, indicating a different rate of gain in each and suggesting a more ready abdominal response to improved nutrition.

The inconstant ratio between the thickness of the fat of different regions, together with the lack of a high correlation between them and the variation of the correlations in different age groups; the conspicuous changes in the amount of fat at various periods, irrespective of change in the nutritional state (such as the prepubertal gains), and the more ready response of the abdominal fat to improved hygienic measures cannot be ignored in any attempt to use the fat fold of the arm as an index of the other subcutaneous fat or the changes in its amount as a measure of a simple nutritional state.

Sex Ratios.—Only the data for the Japanese series are included in chart 15, in order to avoid confusion. A number of noteworthy phenomena were manifested by this race: 1. In weight the girls exceeded the boys after the seventh year. 2. In stature the girls began to exceed the boys by the eighth year, or a year sooner than the white girls surpassed the white boys. While the lead in stature of the white girls over the white boys up to the age of 14 was less than 1 per cent (except for 1.1 per cent at 11), that of the Japanese girls over the Japanese boys ranged up to 6 per cent. 3. In bicristal diameter the Japanese girls attained a greater width than the Japanese boys at the early age of 8 years, whereas 10 to 11 years was the age at which this occurred in the white girls. 4. In arm fat the Japanese girls exceeded the Japanese boys at all ages, and (making allowances for variation due to moderate numbers) the differences increased at each successive

year from 5 to 16. In abdominal fat the same phenomenon was apparent, but the difference between the boys and the girls, in favor of the girls, was still more marked.

Miscellaneous Notes.—Straight legs and a well formed thorax were found in only 29 per cent of the Japanese children. A moderate but noticeable degree of bowed legs and misshapen chest, sometimes both, occurred in 54 per cent—over half. Conspicuous deformity was visible in 16 per cent and severe deformity in 1 per cent. Deformities of the

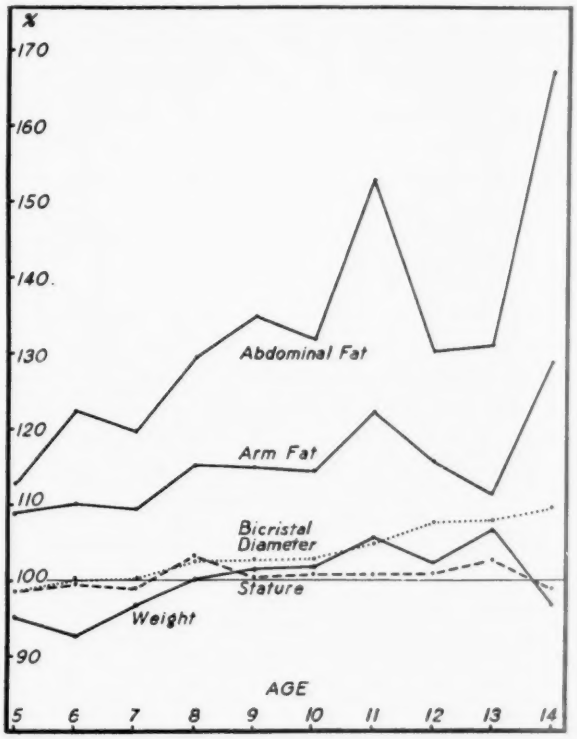

Chart 15.—Sex ratios (girls to boys) for different measurements in Japanese children.

chest, besides the familiar Harrison's groove, and pigeon and funnel breast, frequently were of a character not found commented on in the literature. These were depressions of the general size and shape of a fist, looking much as though the fist had been pushed into a doughy mass that had then hardened into that shape. Sometimes there were two or even three of these deep hollows in various parts of the chest.

Teeth were shockingly bad. By gross inspection perfect sets of teeth were found in only 6 per cent. The milk teeth of the younger

children were in a much worse condition than the permanent teeth of the older children. It is to be regretted that the questionnaire did not include the amount of candy consumed (an indication of replacement of bone-building food). In the examination of the children in the upper social stratum it was not uncommon to have a dainty miss with a beautiful skin of fine texture and coloring open her mouth and show rotten, brown teeth; the contrast was startling. Of the children from 5 to 9 years of age, 4 had twenty carious teeth, many of them mere shells or snags; 3 had nineteen bad teeth; 5 had eighteen decayed teeth; 6 had seventeen teeth with holes; 9 had sixteen teeth with cavities, and 16 had fifteen teeth with caries.

Enlarged tonsils, huge in comparison with those commonly seen in the pediatric clinic, and reddened as well, were plentiful. "Runny" noses were frequent.

Pubescent changes, as indicated by development of the breasts, were present in 17 per cent of the 10 year old girls, in 44 per cent of the 11 years old girls, in 60 per cent of the 12 year old girls and in 100 per cent of those 13 years of age.

The number of children who must have been severely burned when young was remarkable. The scarring extended in most cases over a large part of half the body. Scars from appendectomies were not unusual, and in most instances they were the heavy ones of infection; some scars were even pulled in from the surface of the abdomen by heavy fibrous strands that defied motion. Marks from scarifying to drive out disease were found in symmetrical rows on the back and chest of a small number, mostly older boys.

CONCLUSIONS

Measurements were made of the stature, weight and bicristal diameter of 740 Japanese and 391 Chinese children and, in the case of the Japanese children, of fat folds also. Comparisons are made both with white children of the same region and with Oriental children of similar stock but at different times and in different regions. The social-economic status of the Chinese and Japanese in San Francisco is discussed.

The data for Chinese children strikingly confirm the expected effects on health, growth and development of the unfavorable social-economic situation of this race in San Francisco. That the Chinese children in this city were exceeded in growth in stature by the white children of San Francisco and by the Japanese children of both Los Angeles and San Francisco might be thought to be due to a racial characteristic, but when their stature was found to be surpassed by that of the natives of Canton, from which their families emigrated, and was exceeded by

that of the Cantonese who had settled in Hawaii, the cause must be looked for elsewhere than in race. In weight, the discrepancy is still more evident, especially in comparison with the Hawaiian Cantonese, who at 14 years averaged 20 pounds (7.47 Kg.) heavier. In both height and weight there was none of the prepubertal vigor of growth that was found in both the white children and the Japanese children. The only exception to the lack in the Chinese of the periods of acceleration present in the other races was the pronounced increase in the pelvic width of the Chinese girls of 13.

The stature of the Japanese children of San Francisco has passed that of the Japanese children in California in 1911 but is today surpassed by that of the Japanese children of Los Angeles. On the other hand, the height of the Seattle Japanese of the same stock had nine years ago equaled the present height of the Japanese in Los Angeles. In weight, likewise, the Japanese children in Los Angeles surpassed those of San Francisco. In bicristal diameter, however, the regional influence of Los Angeles seemingly has not proved conducive to as great a pelvic width in the Japanese children there as that attained by their compatriots in central California. In addition, it may be inferred from the data for the Japanese of the South, though this needs corroboration, that the period of prepubertal change there is shorter than that in the North.

The measurements for fat folds on the arm and abdomen showed that the Japanese have a surprisingly thick layer of subcutaneous fat, far surpassing the amount reported by Franzen for white children. The girls showed a prepubertal rise in the thickness of fat folds a year earlier than their prepubertal spurts in stature, weight and width, and the rise was repeated at the ages of 14 and 16. The abdominal fat in both sexes proved to be more ample than the arm fat and apparently was more readily responsive to improved nutrition. An inconstant ratio existed between the arm fat and the abdominal fat, not only from year to year but during the same year. Furthermore, the coefficients of correlation between the arm fat and the abdominal fat are not the same for different age groups and are not considered high enough to warrant the use of the arm fat as a criterion of other subcutaneous fat. The conclusion is reached that the arm fat is neither a sensitive nor a reliable index of change in a simple nutritional state.

The variations between the white children and the Oriental children in stature, weight, bicristal diameter, the ratio of the bicristal diameter to the stature, the amount of subcutaneous fat and periods of acceleration in growth make the use of the tables for prediction of weight which have been calculated from the ratio of the pelvic width to the

stature in relation to the weights of white children of questionable value in predicting the weight of Oriental children.

The Japanese girls were superior to the Japanese boys not merely in weight and pelvic width but, to a large extent, in stature and at all times in the amount of the arm fat and, to a still greater degree, in the amount of the abdominal fat.

Miscellaneous observations on the Japanese children included the high proportion of deformities of the bones, the shockingly bad condition of the teeth, the great prevalence of infections of the upper respiratory tract and of uncommonly large, reddened tonsils and the frequent presence of much scarring, such as heavy appendectomy scars, extensive scars from burns and, in the older boys, symmetrical rows of scarifying burns made to drive out disease.

THROMBOSIS OF THE LATERAL SINUS AND JUGULAR BULB OF NONOTITIC ORIGIN

A NEW DIAGNOSTIC SIGN

SAMUEL KARELITZ, M.D.
NEW YORK

The diagnosis and mode of development of thrombophlebitis of the lateral sinus are usually fairly clear. A septic temperature and chills in the course of purulent otitis media, with or without evidence of mastoiditis, suggest thrombosis either of the lateral sinus or of the jugular bulb. Positive results of blood cultures at this time, as pointed out by Libman,[1] are of great significance and usually indicate that operative treatment is imperative. In all such cases the otitic infection is the start.

In contrast to sinus thrombosis secondary to otitis media, I have observed two children and have been interested in a third child in whose case I believed that the source of sinus thrombophlebitis was not an otitic infection. In one case the condition was due to the spreading of a thrombus from the left lateral sinus to the healthy right side, and in the other two it was retrograde, from thrombophlebitis of the internal jugular vein.

In each instance a peculiar engorgement of the tympanum was observed on the same side as that on which the sinus thrombosis was demonstrated at operation. I believe that in each instance the thrombophlebitis preceded the otitic manifestation, and I therefore wish to present this picture of otitic congestion as a new sign of a new clinical condition, namely, retrograde sinus thrombophlebitis, which I believe must occur on numerous occasions but which has heretofore been observed only at postmortem examination.

CASE 1.—J. N., a 7½ year old girl, had been in excellent physical condition. Of significance in her past history was the fact that at the age of from 4 to 5 years she had repeated and spontaneous painless rupture of the ear drum, with purulent discharge and rapid uncomplicated recovery. A low grade fever usually accompanied this condition.

The present illness started on May 29, 1932, with an earache on the left side and a temperature of 101 F. The condition progressed rapidly, necessitating

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From the Department of Pediatrics, the Mount Sinai Hospital, service of Dr. Béla Schick.

1. Libman, E.: A Further Communication on the Importance of Blood Cultures in the Study of Infections of Otitic Origin, Internat. Otol. Cong., Tr. **9**:127, 1912.

myringotomy, which was carried out by Dr. Samuel Rosen on May 31. A profuse discharge of pus resulted. On June 1 there was a striking postauricular swelling. The child complained of headache on the left side and ocular pain. There was slight blurring of the ocular fundi. On June 2, the fifth day of the illness, Dr. Rosen performed a mastoidectomy. The mastoid was observed to be extensively broken down and was full of pus. The lateral sinus was exposed, and a perisinuous abscess was revealed. The external wall of the sinus was covered with granulations and was adherent to the adjacent bone. It was considered best not to explore the sinus further. Postoperatively the headaches were very severe, and after forty-eight hours the child felt chilly and had a rise of temperature to 104.4 F. A culture of blood taken at this time was sterile. Culture of material taken from the mastoid at the time of operation showed pneumococcus type III. The course of the illness for the next two weeks was characterized by low grade fever, persistent headache and occasional pain in the left eye. The temperature rose to 102 F. on two occasions. On June 18 a sudden rise of temperature, associated with a chill and the obtaining of pneumococcus type III, in blood culture, indicated the necessity of an operation. The left jugular bulb was found to contain a broken-down thrombus. There was also an organized thrombus which extended to the torcular, where free bleeding was obtained.

Postoperatively the temperature remained at about 102 F. The child complained of violent headache in the frontal region and severe headache in the occipital region. There was increasing blurring of the optic nerve head to 5 diopters of papilledema.

On July 3, the thirty-sixth day of illness, the child had a violent convulsive seizure, which lasted for about two hours and involved the right side of the body, leaving right hemiplegia, including paralysis of the right side of the face, and nuchal rigidity. The Babinski, Kernig and ankle clonus reflexes were present. The abdominal reflexes were absent on the right. Lumbar puncture, made during the convulsion, revealed an increase in the spinal fluid pressure and a positive reaction for globulin but only 4 cells. The fluid was sterile on culture.

Lumbar puncture was performed daily for four days and then every other day. The withdrawal of 50 cc. or more of fluid seemed to result in a diminution of the headaches and vomiting. This improvement was further aided by twelve hour periods of continuous intravenous injections of 5 or 10 per cent solution of dextrose in Ringer's solution or in solution of sodium chloride.

On July 12 anomia and urinary incontinence developed. The anomia indicated involvement of the temporal lobe. On July 14 exploratory craniotomy was performed; the left temporal lobe was needled, but no abnormality was found.

On July 23 there were a sudden swelling of the cervical glands on the right side and a peculiar bluish cyanosis of the posterior and inferior portions of the ear drum at the junction with the canal wall. This peculiar discoloration of the drum slowly increased, so that after ten days it had involved most of the right drum. No pain, disturbance in hearing or changes in the canal wall were associated with the condition in the ear.

The possibility of sinus thrombosis on the right side was suggested, but operative intervention was not considered seriously, because of the absence of the usual indicative signs and symptoms. Bilateral temporal hemianopia developed. On July 27 the left temporal lobe of the brain was again explored, and a large communicating abscess was found and drained. Some improvement was noted during the next few days, but after that the child's condition became worse. On August 13 purulent meningitis developed, and on August 15 the child died.

Postmortem examination revealed that the left side of the brain, especially the temporal and frontal lobes, were studded with abscesses, of which some were isolated and others communicated with the abscess cavity which had been drained. Organized thrombosed sinuses were present on the left side. On the right side there was purulent thrombosis of the jugular bulb involving the petrosal and lateral sinuses. There was diffuse purulent meningitis, which was especially severe in the central fissure. The right mastoid was not involved.

The picture of the tympanum which was noted in this child was observed also in the following two cases and in each instance directed my attention to the correct diagnosis.

Case 2.—D. V., a 5 year old girl, became ill on Dec. 26, 1933, with acute tonsillitis and cervical adenitis. Her brother had just recovered from a similar illness, during which a tonsillar abscess was incised and drained, and two other members of the family had similar pharyngeal infections but without suppuration. On December 30 a parapharyngeal abscess on the right was incised. Considerable pus was released, with much relief to the patient. Nevertheless, the cervical glands remained enlarged and tender, and the temperature reached 103 F. the next day.

About forty hours after incision of the abscess there was a severe hemorrhage from the abscess cavity. The hemoglobin content of the blood dropped to 50 per cent. The abscess cavity was packed by Dr. I. Goldman, and the bleeding ceased. An intravenous injection of 300 cc. of 5 per cent solution of dextrose in a solution of sodium chloride resulted in an improvement in the general condition.

On Jan. 2, 1934, chemosis of the right conjunctiva was noted and simultaneously a slight pinkish discoloration of the right tympanum. The temperature remained at 102 F. A transfusion of 200 cc. of blood brought about general improvement. The fever became low grade, and the condition of the cervical glands improved. During this time there seemed to be considerable mucus in the pharynx, and swallowing continued to be difficult, although the abscess swelling had practically disappeared. The conjunctival chemosis increased, and purulent fluid appeared in the anterior chamber, necessitating evisceration of the right eye on January 8. After the operation the child seemed to be improving fairly well, except that the congestion in the right tympanum had increased. The drum became cyanotic and dusky red, and finally the engorgement involved the wall of the canal. The drum appeared thick, but there was no spontaneous pain, and hearing was unaffected.

On February 14 the temperature, after remaining at about 100 F. for several days, suddenly rose to 103 F. No explanation for this change could be found except for the condition of the ear. Myringotomy was therefore performed, but only bleeding resulted. The possibility that thrombosis of the lateral sinus was the cause of the engorgement of the tympanum was therefore suggested. On the two succeeding days the ear drained very little, at first blood and then seropurulent exudate. On February 16, two days after the myringotomy, a chill, a rise of temperature to 105 F. and positive results of blood culture for Streptococcus haemolyticus suggested strongly the presence of thrombosis of the lateral sinus, probably secondary to thrombosis of the internal jugular vein. Involvement of the right mastoid was doubted because of the absence of the usual signs but was considered to be present on roentgenologic examination.

Operation was performed by Dr. Goldman. Except for marked congestion of the mastoid and considerable bleeding, the right ear and mastoid were normal. No

pus, granulation or breaking down of bone was seen. The internal jugular vein above the entrance of the facial vein was found to be collapsed. The jugular bulb and the lateral sinus were completely occluded by thrombus. The usual procedures for this condition were carried out. The child's condition immediately improved, and an uneventful recovery was made.

CASE 3.[2]—J. F., a 12 year old boy, had a sore throat and a low grade fever, followed by swelling of the neck. The swelling became fluctuant and was incised on Sept. 6, 1935. A deep abscess was found and drained, but the temperature remained septic. Blood culture showed Streptococcus haemolyticus. After six days, because of the persistent evidence of sepsis, the cervical wound was reopened, and phlebitis of the internal jugular vein was revealed. The vein was resected. The septic temperature and the results of blood culture remained unaltered after this operation.

At the time it was suggested that the ear should be examined for possible engorgement, as it was thought that there might be a spreading thrombus into the jugular bulb. On September 14 the inferior margin of the right ear drum was found to be bluish red. Hearing was good, and no pain or changes in the wall of the canal were noted. The ear drum became more congested during the next few days. Five days postoperatively the temperature dropped, and it remained normal for six days. The child then seemed well clinically; however, the condition of the ear still suggested the possibility of thrombosis of the jugular bulb and possibly of the lateral sinus. Therefore, it was not certain that the patient was over his illness, as was otherwise indicated. On September 27 the temperature rose to 104 F., and blood culture again showed the presence of Str. haemolyticus. Although the otitic congestion had diminished, these newer findings, together with pain over the right eye, made operative intervention urgent and imperative. At operation, on September 28, Dr. J. Maybaum made the following observation: The right mastoid was normal; the sigmoid sinus close to the jugular bulb was gray and contained a purulent thrombus; the lateral sinus showed marked thickening of its wall and was full of thrombus as far back as 2 cm. from the torcular, where free bleeding was encountered. The phlebitis extended even farther than did the thrombus. There was no bleeding from the petrosal sinus. The temperature dropped several days postoperatively. The child is still under observation.

COMMENT

In case 1, that of the 7 year old child with otitis media and mastoiditis on the left, the diagnosis of sinus thrombosis offered no special problem. Purulent otitis media and mastoiditis, with a subsequent rise in temperature, a chill, a blood culture of pneumococcus type III and some changes in the ocular fundi were noted. At operation the thrombus was found to extend to the torcular, at which point free bleeding was obtained. There was, however, some question at the time of this operation as to whether the wall of the sinus was involved beyond the torcular. It is therefore reasonable that the spread of the thrombus and infection to the right lateral sinus even as far as the

2. Doctors J. Maybaum, A. Winkelstein and H. Neuhof have given me the privilege of presenting this case. A more detailed report will be published later by Drs. Maybaum, Rosen and Goldman.

jugular bulb should have been suspected when sudden and unexplained cervical adenitis on the right side and the peculiar cyanotic appearance of the right tympanum became manifest. The autopsy observations confirmed the earlier impression. It is noteworthy that in spite of the condition on the right side there was no septic temperature or chill. It is possible that, with most of the sinuses occluded, there was a reduced avenue of escape for infected material from the right sinus into the general circulation. The signs and symptoms in this case were sufficiently impressive to warrant a clinical diagnosis of retrograde sinus thrombosis in case 2 before the septic temperature, the chill and the positive results of blood culture were observed.

In case 2, that of a 5 year old child with a pharyngeal abscess, the hemorrhage suggested involvement of a vein of considerable size. The enophthalmos was at first interpreted as being due to bacteremia, but there had been no chill, septic temperature or splenic enlargement up to that time. None of the usual findings of septicemia was present. The simultaneous appearance of a peculiar congestion of the tympanum on the same side as the involved eye was rather striking, but it was believed probably to be due to embolism from the pharyngeal infection. The slow development of the aural changes, the peculiar color of the ear drum, the absence of pain, the persistence of good hearing and finally the absence of pus after myringotomy suggested that the ear was engorged and indicated the presence of sinus thrombosis. Forty-eight hours later a chill, a sudden rise in temperature to 105 F. and positive results of blood culture for Str. haemolyticus were sufficient confirmation of the suspected diagnosis to warrant operative intervention.

In case 3, that of the 12 year old boy who had a cervical abscess after a sore throat, an examination of the ear was not considered to be of importance until the experience in case 2 was recalled. The finding of phlebitis of the internal jugular vein and the continued septicemia after the vein was tied off suggested the possibility of retrograde spread of the thrombosis to the bulb and to the lateral sinus. Only then was the right ear examined for signs of engorgement, which were found and, as in cases 1 and 2, indicated interference with the return circulation from the ear, thereby yielding a clue to the source of the continued septicemia, namely, sinus thrombosis. In view of the present knowledge of this case and the findings in the other two cases, I believe that, confronted by a similar situation, operation via the mastoid for jugular thrombosis would be performed almost immediately after the finding of the peculiar engorgement of the ear on the same side as jugular thrombophlebitis.

Cases 2 and 3 illustrate very clearly how thrombosis of the jugular bulb may be retrograde from an involved internal jugular vein. This suggests that the condition called primary thrombosis of the jugular bulb need not necessarily be secondary to otitis media. In view of the described findings, one can conceive of the possibility that the otitic condition in some cases of primary thrombosis of the jugular bulb is perhaps, as in the present cases, characterized by signs of engorgement.

The anatomic explanation of these otitic findings may be as follows: In cases 2 and 3 thrombophlebitis of the internal jugular vein resulted from the infection of the throat and spread upward against the blood stream into the jugular bulb and the lateral sinus, thus causing venous congestion in the tympanic veins, which empty into the jugular bulb. In like manner there was probably interference with the veins which the bulb receives from the antrum and the mastoid cells and the emissary vein from the mastoid which empties into the sigmoid sinus. In case 3 the late diminution of the congestion may have been due to the setting up of a collateral circulation with the peripheral circulation. In case 1 the thrombophlebitis spread from the left lateral sinus to the right, occluding the right lateral sinus and the jugular bulb and leading to tympanic congestion in the same manner as in cases 2 and 3.

CONCLUSIONS

Retrograde thrombophlebitis of the jugular bulb and the lateral sinus may occur and can be recognized clinically. In two cases it was secondary to thrombophlebitis of the internal jugular vein and in the third to a retrograde spread from the diseased left lateral sinus to the healthy right lateral sinus and jugular bulb.

A new finding is described, which consists of painless involvement of the ear; bluish or dark bluish-red engorgement [3] of the drum, starting at the periphery and gradually involving the entire drum and even the adjacent parts of the canal wall; persistence of good hearing; absence of changes in the canal wall other than that of congestion, and absence of signs of involvement of the mastoid, except possibly slight tenderness due to congestion. This picture, either with or without simultaneous involvement of the cervical glands of the same side, should suggest the presence of thrombosis of the jugular bulb or of the lateral sinus or both. When chills, a septic temperature and positive results of blood culture for hemolytic streptococci are observed along with the

3. In discussing the subject-matter of this paper, Dr. I. Friesner referred to the case of a man in whom thrombosis of the lateral sinus developed after involvement of the cavernous sinus. In this case he recalls seeing engorgement of the tympanum such as is here described.

otitic findings, surgical exploration of the sinuses of the affected side is indicated.

NOTE.—Since submitting this paper for publication I have learned of six more cases of retrograde thrombophlebitis of the lateral sinus and jugular bulb.

In two of these cases the condition was diagnosed at autopsy, and in the other four a diagnosis was made clinically after this paper was read at the Nov. 6, 1935, meeting of the New York Pediatric Club.

In the four cases in which a clinical diagnosis was made there was a history of acute pharyngitis, cervical suppuration (probably of broken-down glands in the region of the internal jugular vein) and bacteremia. In two of these cases otitic engorgement was noted; in the other two the condition of the ear was not noted, the correct diagnosis having been suggested by the finding of thrombophlebitis of the internal jugular vein on operation for the cervical suppuration.

CONGENITAL CARDIAC ANOMALIES IN INFANTS

REPORT OF FIVE CASES—(1) ACCESSORY VENTRICLE, (2) TETRALOGY OF FALLOT WITH RIGHT AORTIC ARCH AND REDUNDANT LEFT DUCTUS ARTERIOSUS, (3) TETRALOGY OF FALLOT WITH ANOMALOUS BAND IN RIGHT AURICLE, (4) COMPLETE TRANSPOSITION OF ARTERIAL TRUNKS AND (5) DOUBLE DEFECT OF VENTRICULAR SEPTUM

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We are presenting the clinical and pathologic observations made with regard to five infants with unusual congenital cardiac anomalies.

REPORT OF CASES

CASE 1.—M. P. was admitted to the hospital on Nov. 22, 1932, and was discharged on November 30. The infant was 6 weeks old on admission and had a history of cyanosis of four days' duration. The infant was delivered at term by forceps. The birth weight was 8 pounds and 3 ounces (3,742 Gm.). The infant had been well until four days before admission and was fairly well nourished but dyspneic. There was an infection of the upper respiratory tract, and the lungs were dull to percussion, especially over the upper lobe of the right lung, with diminished breathing. The heart was normal. The infant was placed in an oxygen tent for three days; removal from the tent caused no respiratory embarrassment. On discharge slight cyanosis and dyspnea were present. The blood count showed 3,070,000 erythrocytes, 80 per cent hemoglobin and 10,800 leukocytes, with 36 per cent polymorphonuclears and 62 per cent lymphocytes. Examination of the urine revealed no abnormality. The Mantoux test was negative. Roentgen examination showed enlargement of the heart, chiefly of the left ventricle (fig. 1 *B*). A diagnosis of congenital cardiac disease was made.

After discharge the infant was well for about a week, after which it began to have attacks of dyspnea, which gradually grew worse. The infant was readmitted to the hospital on December 12. Examination showed enlargement of the heart to the right and to the left, with a rapid regular beat and no murmurs. There was exaggerated breathing in the upper lobe of the left lung, and a few scattered râles were noted at the bases of the lungs. The infant was placed in an oxygen tent but grew rapidly worse and died the same day.

From the Pediatric Service and the Pathologic Laboratory of the New York Nursery and Child's Hospital.

Specimens were exhibited, and the first two cases were presented, before a meeting of the International Association of Medical Museums, New York, April 17, 1935.

Complete autopsy,[1] except of the head, was performed forty-five minutes post mortem. Only the pertinent observations are given here. The body was well developed and well nourished, measuring 59 cm. in length. The diaphragm was at the level of the sixth rib on both sides. The peritoneal cavity contained about 20 cc. of clear yellow fluid; the retroperitoneal tissues showed moderate edema. There was no pleural effusion.

The heart was markedly enlarged and dilated, weighing 90 Gm. (about twice normal); the apex was bifid. The pericardium was glistening. Because of rota-

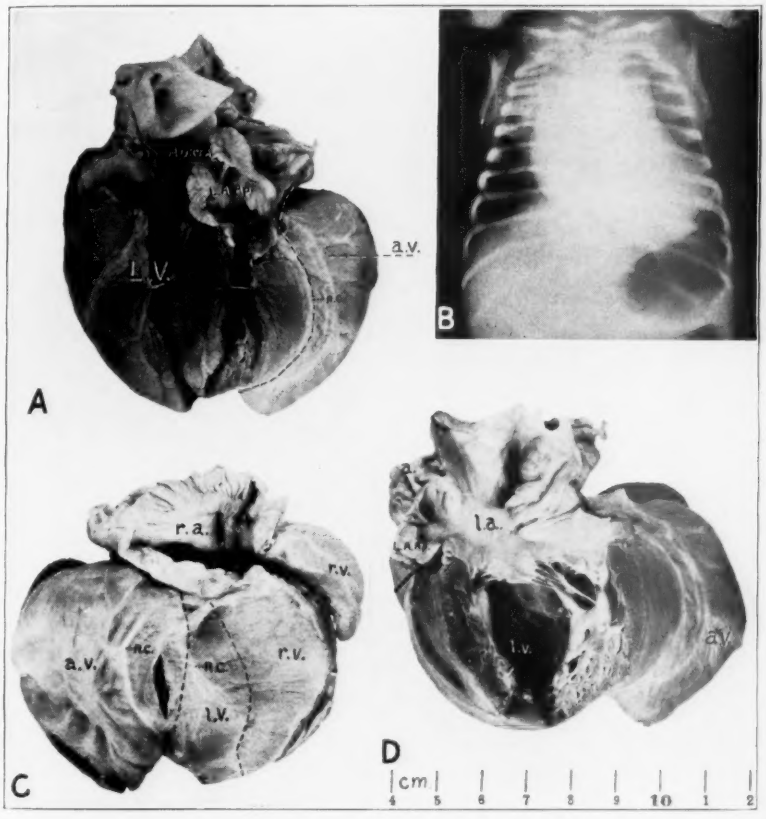

Fig. 1 (case 1).—*A*, anterior aspect of the heart; *B*, roentgenogram of the chest at 6 feet (183 cm.); *C*, posterior aspect, and *D*, left auricle and ventricle. The broken lines indicate the boundaries of the chambers. *a.* indicates aorta; *a.v.*, accessory ventricle; *l.a.*, left auricle; *L.A.Ap.*, left auricular appendage; *l.v.*, left ventricle; *r.a.*, right auricle; *R.C.*, right coronary artery; *r.v.*, right ventricle.

tion to the right the left ventricle formed the anterior surface of the heart to the right of the bifid apex. To the left of the bifid apex was located an accessory ventricle (fig. 1 *A* and *C*). The right auricle was dilated but otherwise normal. The foramen ovale, 5 mm. in diameter, though patent, was functionally closed.

1. The autopsy in all the cases was performed by Dr. F. A. Hemsath.

The tricuspid valve was normal. The right ventricle had a normal structure, but its transverse diameter was greatly decreased because of bulging of the ventricular septum. The pulmonary valve, artery and branches were normal. The ductus arteriosus was closed. The left auricle was moderately dilated, and the pulmonary veins entered in normal relation (fig. 1 *D*). The bicuspid valve was normal. The papillary muscles of the posterior cusp arose conjointly from a broad, thickened base (fig. 1 *D*). The left ventricle, which was greatly hypertrophied and dilated, was lined by glistening, white, thickened endocardium, the thickening being most pronounced at the base. The aortic valve was normal, as was the origin of the coronary arteries. Behind the posterior cusp of the mitral valve (fig. 2 *C*) a

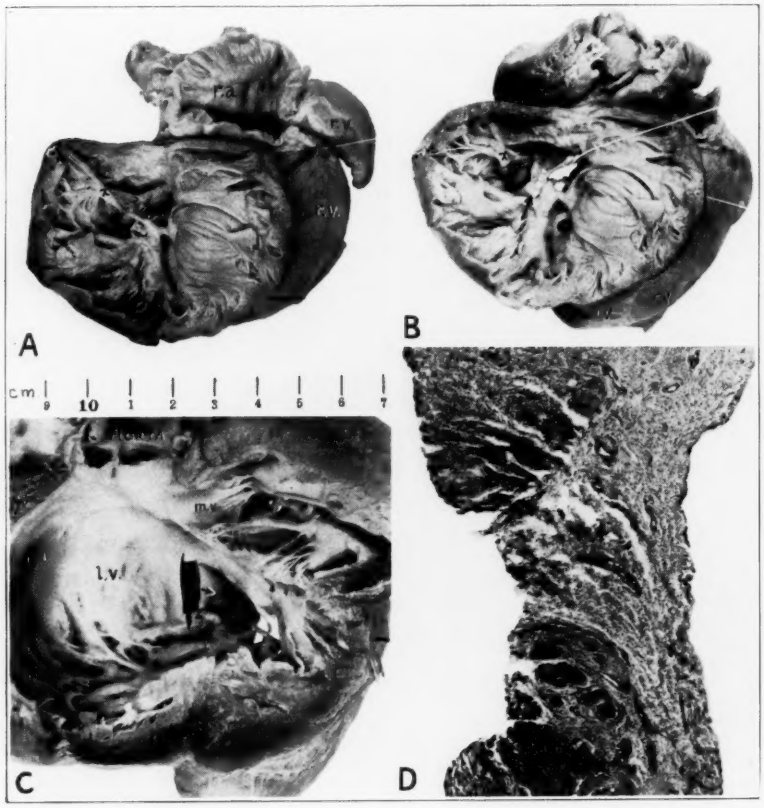

Fig. 2 (case 1).—*A* and *B*, the interior of the accessory ventricle. The muscular cord, *x*, has been reflected in *B*; the arrow points to a valvelike membrane, and the dark probe is inserted in a small pocket to which the vertical incision leads in figure 1 *C*. *C*, the left ventricle with the posterior mitral cusp reflected by an incision behind its papillary muscle, exposing the large opening into the accessory chamber (arrow) and the three small openings (small arrows). *D*, low power photomicrograph of a section of the wall from the apex of the accessory ventricle. On the right is thickened pericardium without leukocytic infiltration. *l.v.* indicates left ventricle; *m.v.*, mitral valve; *r.a.*, right auricle; *r.v.*, right ventricle.

roughly quadrilateral opening, measuring 15 by 18 mm., gave access to a large accessory ventricle (fig. 2 *A* and *B*). The posterior wall of the opening was smooth and directly continuous with the posterior wall of the left ventricle. The superior border was formed by the smooth inferior aspect of the posterior portion of the mitral ring. The anterior and inferior borders were trabeculated. Three minute communications also were present directly beneath the attachment of the posterior cusp of the mitral valve. The accessory chamber measured 55 mm. in length and 35 mm. in width, and its wall varied in thickness from 2 to 7 mm. The glistening muscular walls of the chamber showed a trabecular structure, and in the anterior superior portion was a thin membranous valvelike structure with chordae tendineae (fig. 2 *B*). The anterior surface of the accessory ventricle was smooth and muscular and in no way different in appearance from the adjacent surface of the left ventricle, from which it was separated by a sulcus (fig. 1 *A* and *D*). The posterior surface showed several pale, glistening linear depressions, which produced a bossed surface and were caused apparently by a thin fibrous wall and underlying trabecular structure. Sulci on the posterior surface outlined the position of a small tertiary pocket. Microscopic examination of tissue from the apex of the accessory ventricle (fig. 2 *D*) showed a thickened, fibrous pericardium with the myocardium deficient in the thinnest portions. There was no leukocytic infiltration.

The left coronary artery showed a normal descending branch but a short circumflex branch. The right coronary artery passed completely across the posterior surface of the heart in the coronary sulcus from right to left, giving off three large descending branches (fig. 1 *A* and *C*). The first of these descended along the posterior longitudinal sulcus. The second supplied the posterior portion of the accessory ventricle. The terminal portion coursed to the anterior surface and passed down the sulcus between the left and the accessory ventricle. Thus the accessory ventricle was supplied entirely by the right coronary artery.

The following measurements were noted: total length, 70 mm.; diameter of the base, 85 mm.; circumference of valves: tricuspid, 53 mm.; pulmonary, 30 mm.; bicuspid, 50 mm.; aortic, 27 mm.

The lungs showed moderate congestion at the bases. The right lung weighed 39 Gm. and the left 30 Gm. The paratracheal lymph nodes were enlarged to 5 mm. in diameter.

The other organs were normal, and there were no other congenital anomalies.

The anatomic diagnosis included pulmonary congestion, slight ascites and the following congenital cardiac anomalies: large accessory ventricle, patency of the foramen ovale and cardiac hypertrophy and dilatation.

Comment.—A careful search of the literature has failed to show the report of any anomalies that at all resembled this one. The structures reported as diverticula consisted of muscular finger-shaped projections from the cardiac apex, extending to or through the diaphragm and usually associated with umbilical hernia. They are explained as due to amniotic adhesions. Koller-Aeby [2] reported one case and collected reports of four others from the literature. Mahrburg [3] has added a

2. Koller-Aeby, H.: Ein angeborenes Herzdivertikel in einer Nabelschnurhernie, Arch. f. Gynäk. **82**:185, 1907.

3. Mahrburg, S.: Ueber einen Fall von angeborenem Divertikel des Herzens, Virchows Arch. f. path. Anat. **277**:498, 1930.

case. The only case in an adult has been reported by Alekseeff and Khaimovitch.[4] The case here reported is unique as to the location, size, gross structure and clinical significance of the anomaly, with death resulting from circulatory failure. We consider this defect as an abortive developmental attempt toward the formation of a double heart and as due to an excess of cardiac anlage. In a sense it is analogous to the formation of accessory digits, and we have therefore called it an accessory ventricle.

Case 2.—R., a girl, was admitted to the hospital on Nov. 26, 1933, the day of birth, and died on Jan. 30, 1934. She was born prematurely, and the birth was precipitate. The mother had glomerular nephritis. On the eighth day there was an attack of cyanosis during feeding. A mixture of oxygen and carbon dioxide was administered, but it did not give relief. Gavage was started, but the cyanosis became worse. Feeding was again resorted to. The cyanosis improved slowly but recurred with exertion. When the infant was 1 month old a systolic murmur was heard in the third and fourth left interspaces, and this persisted until death.

At the age of 5 weeks the infant began to vomit. Several days later diarrhea began. The infant became dehydrated and was given dextrose solution by the continuous drip method. Starvation treatment was then started. The infant became edematous, and the administration of dextrose was stopped. The serum protein content was 4.06 Gm. per hundred cubic centimeters of blood. Food was given slowly, but after a few days diarrhea again became manifest, and the administration of dextrose by the continuous drip method was again tried. Edema recurred, with a serum protein content of 3.4 Gm. Râles were heard at the base of each lung on the day before death, which occurred when the infant was 2 months and 3 days old.

Roentgen examination on December 7 showed no enlargement of the heart. Roentgenograms taken two days later suggested an abnormal location of one or more of the great vessels.

A complete autopsy was made, except of the head, twelve hours post mortem. The infant was well developed and well nourished and measured 49 cm. in length. The thymus was small. The right and left lungs weighed 29 and 26 Gm., respectively, and were moderately congested. Microscopic examination showed serous exudate and in some portions a few polymorphonuclear cells in the alveoli. The liver weighed 140 Gm. and showed extensive fatty infiltration. The spleen, weighing 8 Gm., showed congestion and moderate hemosiderosis. The adrenal glands and the kidneys showed cloudy swelling. The small intestine was intensely congested, and shallow ulcerations were present in the jejunum.

The long axis of the heart was located in the horizontal plane and measured 58 mm. The transverse diameters at the base measured 30 and 40 mm. The right auricle and ventricle were dilated. The foramen ovale was patent, measuring 4 mm. in diameter, but showed good functional closure. The tricuspid valve was normal. The left superior portion of the ventricular septum showed a defect 6 mm. in diameter (fig. 3 *A*). The apex of the pulmonary conus was small and gave off a pulmonary artery 4 mm. in diameter, which was completely shut off from the conus by a thin transparent membrane. The left auricle, bicuspid valve and left ventricle showed a normal structure, aside from poorly developed

4. Alekseeff, M. V., and Khaimovitch, D. I.: Extremely Rare Case of Diverticulum of the Heart in an Adult, Russk. klin. **13**:83, 1930.

trabeculae in the ventricle. The dextroposed aorta overrode the septal defect largely to the right. The aortic arch passed to the right of the trachea, displacing the superior vena cava to the right and giving off in succession a left transverse branch, the right common carotid artery and the right subclavian artery (fig. 3 *A*). The left transverse branch passed in front of the trachea, giving off a branch to the thyroidea ima immediately, the left common carotid artery 4 mm. from its origin and the left subclavian artery 7 mm. from its origin. It continued in the form of a semicircle, the diameter of which was 30 mm., to terminate in the pulmonary artery at the point of its bifurcation. The internal diameter of this

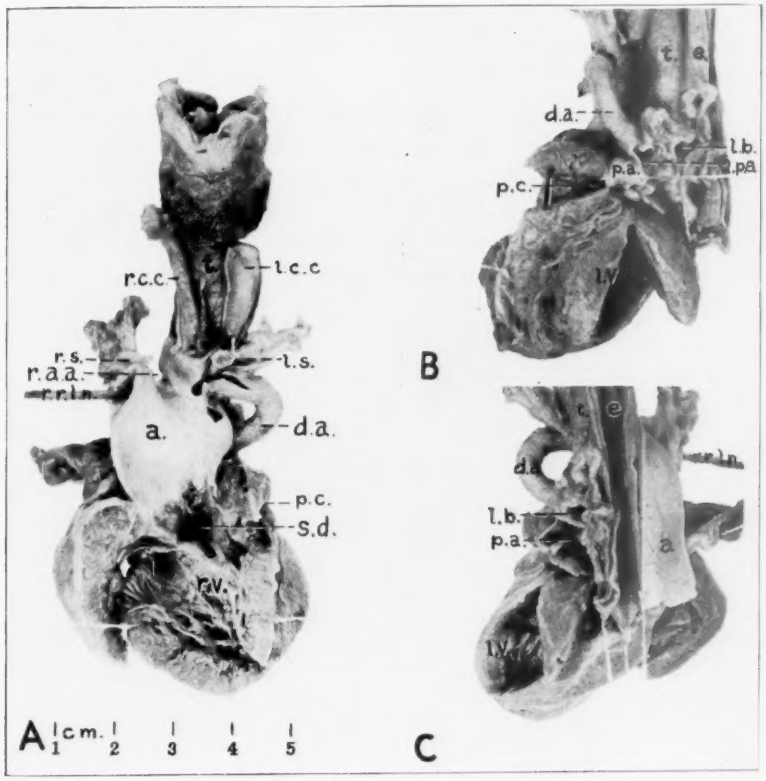

Fig. 3 (case 2).—*A*, anterior view; *B*, left lateral view; *C*, posterior view of the heart. *a.* indicates aorta; *d.a.*, redundant ductus arteriosus; *e.*, esophagus; *l.b.*, left bronchus; *l.c.c.*, left common carotid artery; *l.s.*, left subclavian artery; *l.v.*, left ventricle; *p.a.*, pulmonary artery; *p.c.*, apex of pulmonary conus; *r.a.a.*, right aortic arch; *r.c.c.*, right common carotid artery; *r.r.l.n.*, right vagus nerve; *r.s.*, right subclavian artery; *r.v.*, right ventricle; *s.d.*, defect of the ventricular septum; trachea.

anomalous vessel was 4 mm., and it gave off no branches between the subclavian and the pulmonary vessel (fig. 3 *A* to *C*). The descending aorta, after passing behind the right bronchus, inclined to the left and occupied its normal position in the abdomen. It gave off no abnormal vessels, and there was no trace of any vessel completing the left aortic arch. The right recurrent laryngeal nerve passed

beneath the right aortic arch. The left vagus nerve passed in front of and beneath the first portion of the anomalous vessel, giving off its recurrent branch, and passed to its normal position behind the left bronchus.

The origin of the coronary arteries was normal. The right one was small and consisted of only a circumflex portion which terminated in a branch descending along the midportion of the right ventricle. The left coronary artery was larger than normal and furnished both the anterior and the posterior descending branch, which passed along the corresponding longitudinal sulci.

The anatomic diagnosis included pulmonary congestion, with early pneumonia, enteritis, fatty degeneration of the liver and the following congenital cardiac anomalies: the tetralogy of Fallot, with pulmonary atresia, a right aortic arch, a redundant and anomalous course of the patent left ductus arteriosus and an anomalous course of the coronary arteries.

Comment.—Abbott [5] has reported a case in which there was a right aortic arch, with a patent right ductus arteriosus, in the presence of pulmonary atresia and a septal defect. Anatomically our case is of interest because of the circuitous, nonbranching course of the anomalous vessel and the absence of even a fibrous cord completing the left aortic arch. The fact that the anomalous vessel terminated at the bifurcation of the pulmonary artery indicates that the terminal portion was formed from the embryonal sixth left aortic arch. Whether the proximal portion had the same origin or included also the fourth left embryonal arch seems a matter of speculation. We have not been able to reconcile the position of the left recurrent laryngeal nerve with any theory of evolution of the aortic arch. Anomalies of the embryonal aortic arches may well have been present.

Clinically the case was of interest because of the wide shadow of the upper mediastinum disclosed by the roentgenogram.[6]

CASE 3.—M., a boy, was admitted to the hospital at the age of 11 days, on Jan. 2, 1934, and died on January 18. There was a history of cyanotic attacks during the week preceding his admission. He had no fever but was markedly cyanotic. Percussion did not indicate enlargement of the heart, but a systolic murmur was heard over the third left intercostal space. There were occasional coarse râles in the lungs. A diagnosis of congenital cardiac disease or cerebral hemorrhage was made. The child was placed on a diet of evaporated and breast milk. On the sixth day diarrhea developed, and dehydration set in. The child was starved and then given dextrose solution by continuous drip method for forty-eight hours. The condition of the stools improved, and the child was given a diet of curd protein milk. The dehydration continued, however, in spite of infusions of dextrose solution and hypodermoclyses of solution of sodium chloride. The course was continually downward, with frequent cyanotic spells, until death occurred, at the age of 27 days. On January 2 the blood count showed: erythrocytes, 4,380,000; hemoglobin, 105 per cent; leukocytes, 6,900; polymorpho-

5. Abbott, M. E., in Blumer, G.: Bedside Diagnosis, Philadelphia, W. B. Saunders Company, 1928, vol. 2, p. 500.

6. The roentgenograms of this patient were shown by Dr. H. C. McIntosh before a meeting of the New York Roentgen Society and subsequently were misplaced, incident to the closing of the New York Nursery and Child's Hospital.

nuclears, 59 per cent, and lymphocytes, 39 per cent. Examination of the urine revealed no abnormality. On January 15 chemical analysis of the blood showed: nonprotein nitrogen, 31.6 mg. per hundred cubic centimeters of blood; protein, 4.85 Gm., and chlorides, 345.7 mg. (as sodium chloride, 569.9 mg.). The carbon dioxide-combining power was 41 volumes per cent. The roentgenographic report (fig. 4 *B*) by Dr. H. C. McIntosh was as follows: "The heart is not enlarged, as gaged by the tota! thoracic width. However, its contour shows a flattened pear shape, which is somewhat abnormal. There is inconclusive evidence of congenital cardiac disease."

Autopsy, performed twenty-two hours post mortem, showed a moderately developed, poorly nourished white boy, measuring 48 cm. in length. Five cubic centimeters of clear pink fluid was present in each pleural cavity. The thymus was normal in size and structure.

The heart showed dilatation of the right auricle but was not otherwise enlarged. The apex was bifid. In the right auricle the orifices of the superior and the inferior vena cava and the coronary sinus were normally located, and that of the coronary sinus was slightly dilated. The fossa ovale measured 14 mm. in diameter. The septum primum showed complete peripheral attachment, and five fenestrae, averaging 3 mm. in diameter (together with a number of smaller ones), were

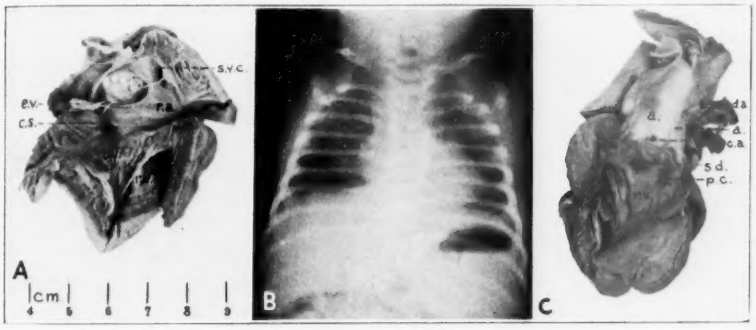

Fig. 4 (case 3).—*A*, the right auricle and ventricle; *B*, roentgenogram of the chest; *C*, the right side of the heart, showing the pulmonary conus. *a.* indicates aorta; *c.a.*, left coronary arteries; *c.s.*, orifice of coronary sinus; *d.a.*, ductus arteriosus; *e.v.*, eustachian valve; *p.c.*, apex of pulmonary conus; *r.a.*, right auricle; *r.v.*, right ventricle; *s.d.*, defect of ventricular septum; *s.v.c.*, orifice of superior vena cava; *t.v.*, tricuspid valve.

present in its upper portion (fig. 4 A). A thin fibrous band crossed the chamber, arising by a triple origin from the free border of the eustachian valve to be inserted in a thin crescentic vertical band located along the left margin of the orifice of the superior vena cava. The thebesian valve was poorly developed. Further description of the heart will be abbreviated. The structure of the apex of the right ventricle suggested aneurysmal dilatation (fig. 4 *C*), the myocardium measuring 1 mm. in thickness. The defect of the left superior portion of the ventricular septum opened from the conus pulmonalis and measured 9 mm. in diameter. The aorta, with a normal valve, measuring 30 mm. in circumference, overrode the defect. The stenosed pulmonary valve, located behind and to the left of the aorta, measured 12 mm. in circumference and showed three small cusps of equal size. The left side of the heart was poorly developed. The wall

of the right ventricle measured 5 mm. in thickness; that of the left, 6 mm. The ductus arteriosus admitted a probe 2 mm. in diameter. The right coronary artery was small, and its distribution was restricted. The left coronary artery showed a dual origin and furnished both the anterior and the posterior longitudinal vessel.

The left lung weighed 28 Gm.; the right, 32 Gm. The right lung was divided into four lobes and the left into three. Except for the anterior margins, the lung tissue was dark and firm. Microscopic examination showed massive polymorphonuclear infiltration of the alveoli and interstitial tissue, with abscess formation.

The liver weighed 116 Gm. and showed slight passive congestion and cloudy swelling. The gallbladder was crossed by a peritoneal band, producing an hourglass shape.

The peritoneum showed several abnormal bands, which caused stenosis at the junction of the descending and the horizontal portion of the duodenum, with dilatation of the first portion of the duodenum, and at the splenic flexure of the colon, proximal to which point the intestine was dilated. The mesocecum and the ascending mesocolon were 30 mm. in length, and the cecum and the ascending colon consequently were located at the midline. The cecum was aplastic, being smaller in diameter than the terminal portion of the ileum. Well formed, soft stool was present in the descending colon. No portion of the intestine was inflamed. The abdominal organs were not otherwise remarkable. The right hypogastric artery was represented by a thin fibrous cord; the left was increased in size.

Examination of the skull revealed no abnormality aside from a rudimentary development of the posterior clinoid processes.

The anatomic diagnosis included pneumonia, pleural effusion and the following congenital anomalies: (*A*) heart—stenosis of the pulmonary valve and artery, a defect of the ventricular septum, dextroposition of the aorta, dilatation of the right auricle, fenestration of the septum primum, slight patency of the ductus arteriosus, an anomalous band in the right auricle, an abnormal course of the coronary arteries and aneurysmal dilatation of the apex of the right ventricle; (*B*) vessels—atresia of the right hypogastric artery; (*C*) gastro-intestinal tract—a mobile, aplastic cecum and stenosis of the gallbladder, duodenum and splenic flexure of the colon caused by peritoneal bands; (*D*) skull—aplasia of the posterior clinoid processes.

Comment.—As in case 2, we are not commenting on the tetralogy of Fallot (pulmonary stenosis or atresia, with a defect of the interventricular septum, dextroposition of the aorta and hypertrophy of the right ventricle), which is a not uncommon anomaly and presented no unusual features in these cases.

The origin and termination of the anomalous band in the right auricle indicate, we believe, that it was a remnant of the free border of the right valve of the sinus venosus. The fenestrae of the septum primum suggest that fenestration of the right valve of the sinus venosus may have prevented the normal retraction of this tissue remnant. Yater [7] discussed such bands (network of Chiari) in detail and added reports of four cases in which they were present to the twenty-two

7. Yater, W. M.: Variations and Anomalies of the Venous Valves of the Right Atrium of the Human Heart, Arch. Path. **7**:418 (March) 1929.

in the literature. The bands are clinically significant, because thrombi may form on them, causing fatal embolism.

CASE 4.—F. R., a 3 month old boy, was admitted to the hospital on Oct. 7, 1932, and was discharged on October 11. He had been cyanotic since birth. On admission he appeared underdeveloped. Cyanosis was continuous but was aggravated by crying. There was no respiratory distress. The fontanels and sutures were wide open; craniotabes was marked, and the veins of the scalp were prominent.

Percussion showed that the heart was enlarged to the right and to the left. A loud, rough systolic murmur was heard over the precordium, being most marked over the left second and third interspaces, and was transmitted to the cervical vessels. It was heard both in front and in back. The lungs were clear. The Mantoux and Wassermann tests were negative. The red blood count showed: erythrocytes, 5,900,000; hemoglobin, 115 per cent; leukocytes, 8,700; polymorphonuclears, 47 per cent, and lymphocytes, 52 per cent. Urinalysis revealed no abnormality. Roentgenograms showed an enlargement of the heart.

A diagnosis of congenital cardiac disease was made.

The infant was readmitted on December 29 and was discharged on Jan. 12, 1933.

While at home the infant took the formula poorly, and the cyanotic attacks continued. On readmission there was an infection of the upper respiratory tract but no fever. There was evidence of rickets. The infant was put on a whole milk formula and began to gain in weight. He was discharged improved. The blood count showed: erythrocytes, 4,900,000; hemoglobin, 83 per cent; leukocytes, 8,200; polymorphonuclears, 33 per cent, and lymphocytes, 63 per cent. Urinalysis revealed no abnormality. Chemical analysis showed: calcium, 9.92 mg. per hundred cubic centimeters of blood, and phosphorus, 5.52 mg.

The infant was admitted for a third time on January 17 and died on January 26. He was cyanotic but not dyspneic. The results of physical examination were similar to those obtained on previous admissions. The day after admission the temperature rose to 104 F., and the respiration became rapid and dyspneic, with an occasional grunt. The lungs were normal. On the fourth day cervical rigidity and a Brudzinski sign were noted. A spinal tap gave clear fluid under increased pressure, but the cytology was normal. A week after the beginning of the fever, fine râles were first heard throughout the right side of the chest. The child's condition grew worse, and the fever continued until death occurred, at the age of 6 months.

On January 17 a blood count showed: erythrocytes, 5,100,000; hemoglobin, 86 per cent; leukocytes, 11,200; polymorphonuclears, 54 per cent, and lymphocytes, 46 per cent. On January 20 a blood culture gave positive results for nonhemolytic streptococci. On January 25 the blood culture was sterile. The urine was normal. A roentgenogram, made on January 19 (fig. 5 *A*), showed the transverse width of the heart to be 7 mm. greater than on the first admission. The lungs were clear.

Autopsy, performed twenty-two hours post mortem, showed a well developed, well nourished, white boy, measuring 64 cm. in length. The anterior fontanel was wide open. The diaphragm was at the level of the fifth rib on each side. The chondral portion of the right fourth rib was forked between the sternum and the epiphysis. There was no free fluid in the serous cavities.

The heart showed generalized enlargement, with the left border in the anterior axillary line, displacing the lung. Its total length was 96 mm., and the diameters at the base were 50 and 65 mm. The external configuration was normal, aside from dilatation of the right auricle and the reversed position of the arterial trunks.

A patent foramen ovale, 4 mm. in diameter, was well protected by the free margin of the septum primum; otherwise the dilated right auricle had a normal structure. The tricuspid valve was normal and measured 54 mm. in circumference. The left superior portion of the ventricular septum showed a smooth-bordered oval defect, measuring 12 by 10 mm., which afforded a free communication between the pulmonary conus and the aortic vestibule (fig. 5 *C* and *D*). The pulmonary conus was otherwise normally developed, and the aorta arose from the position normally

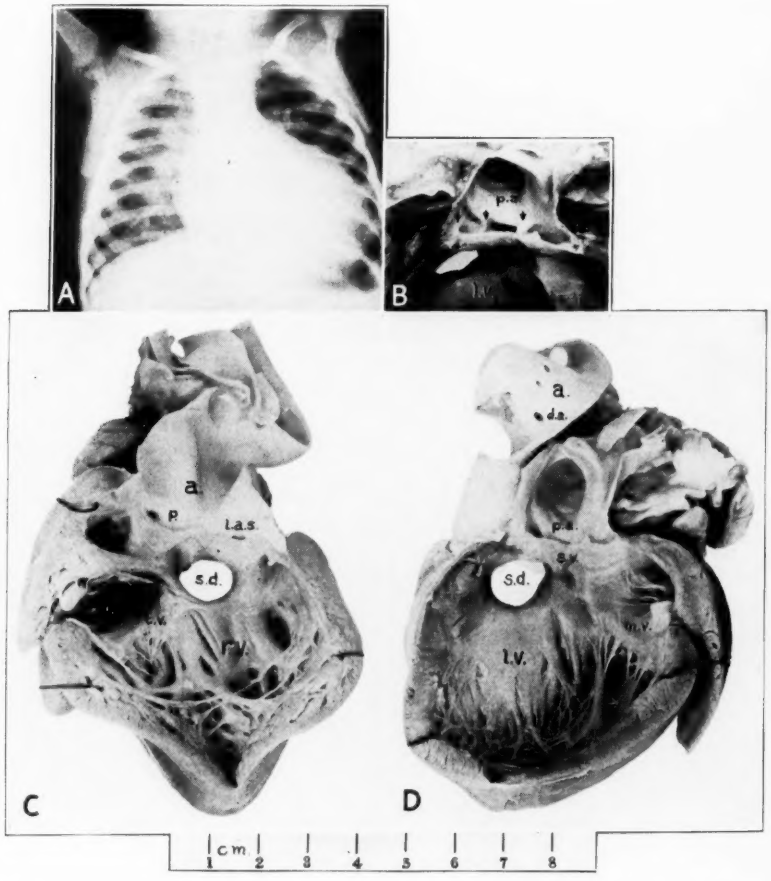

Fig. 5 (case 4).—*A*, roentgenogram of the chest; *B*, the bicuspid valve of the pulmonary artery from above. The middle one of the three arrows points to the abortive pillar of the valve. *C*, the right ventricle; *D*, the left ventricle. *a.* indicates aorta; *d.a.*, ductus arteriosus; *l.a.s.*, left anterior sinus of Valsalva; *l.v.*, left ventricle; *m.v.*, mitral valve; *p.*, posterior sinus of Valsalva; *p.a.*, pulmonary artery; *r.v.*, right ventricle; *s.d.*, defect of ventricular septum; *s.v.*, pulmonary semilunar valves; *t.v.*, tricuspid valve.

occupied by the pulmonary artery. The membranous tissue, seen in figure 5 *C*, between the right superior border of the septal defect and the converging convex

margins of the posterior and left anterior cusps of the aortic valve, consisted of the "external" aspect of the posterior pulmonary sinus of Valsalva. The aortic valve and the branches of the aortic arch were normal. On the left side the pulmonary veins, the auricle and the mitral valve were normal. The mitral valve was 47 mm. in circumference. The pulmonary artery came off the left ventricle from the position normally occupied by the aorta. The pulmonary valve was bicuspid and stenosed, and its cusps were glistening, translucent and thickened. Its circumference measured 29 mm., in contrast to that of the aortic valve, which was 45 mm. The posterior cusp was wider than the anterior. The posterior sinus of Valsalva showed a vertical ridge representing abortive pillars of the valve (fig. 5 *B*). Microscopic examination of the cusps showed that the thickening was due to embryonal tissue. Both the aorta and the pulmonary artery showed orifices of the ductus arteriosus, which measured 2 and 1 mm. in diameter, respectively. The ductus measured 12 mm. in length, and its lumen at the midportion admitted a probe with a diameter of only 0.5 mm. The reversal of the normal degree of trabecular development in the two ventricular chambers is well shown in the illustrations. The endocardium was pale throughout and the myocardium firm.

The coronary arteries, in brief, showed the following anomalies: The left coronary artery showed a normal origin, but its circumflex branch was absent, and a large additional descending branch passed down to the left of the anterior longitudinal sulcus. The right coronary artery arose from the posterior sinus (fig. 5 *C*) and gave off a left circumflex branch which passed anteriorly to the left as far as the anterior longitudinal sulcus. Its posterior circumflex branch passed beyond the posterior longitudinal sulcus and descended at an angle divergent from it to supply the left posterior portion of the left ventricle.

The right lung weighed 63 Gm. and the left 45 Gm. An area of consolidation, 2 by 5 cm., was present in the lower lobe of the left lung. Microscopically it showed diffuse alveolar and interstitial polymorphonuclear infiltration. The right lung showed circumscribed dark, firm areas about the hilus in all the lobes. Both lungs showed peribronchial thickening, with polymorphonuclear and round cell infiltration. On the left, polymorphonuclear exudate was present in the bronchi.

There were no changes of importance in the other organs. Examination of the head was not permitted.

The anatomic diagnosis included bilateral bronchial and diffuse pneumonia and the following congenital anomalies: (*A*) heart—complete transposition of the arterial trunks, a defect of the intraventricular septum, a bicuspid pulmonary valve with slight stenosis, a patent foramen ovale, a minute patency of the ductus arteriosus, persistence of the embryonal thickening of the cusps of the pulmonary valve, cardiac hypertrophy and dilatation and an anomalous origin and course of the coronary arteries; (*B*) osseous system—forked chondral portion of the right fourth rib.

Comment.—While this type of cardiac anomaly does not represent a great rarity, the large size of the septal defect was unusual. Abbott [8] listed thirty-six cases of complete transposition of the arterial trunks with a septal defect in her analysis of one thousand cases of congenital abnormality of the heart. The patent foramen ovale in this case permitted passage of blood only from the right to the left auricle. In

8. Abbott, M. E., in Nelson Loose-Leaf Living Medicine, New York, Thomas Nelson & Sons, 1931, vol. 4, p. 228.

a heart with normally located arterial trunks it is customary to speak of this as functional closure; but when the arterial trunks have been transposed so that the (systemic) pressure is higher on the right side, this one way passage through the foramen is undoubtedly in use, and the term is therefore not applicable.

The deformity of the right fourth rib in this case is an example of the type discussed by Shain.[9]

Case 5.—D. A., a white girl, the younger of twins, was born in the New York Nursery and Child's Hospital on Sept. 10, 1932. Delivery was by breech extraction. The weight at birth was 8 pounds and 1 ounce (3,657 Gm.), which was 1½ pounds (227 Gm.) more than the weight of her twin. While in the hospital she was not cyanotic or dyspneic, and no murmurs were heard. When 20 days old, the infant was brought to the outpatient clinic for routine attention. The mother had recently noticed that the infant stopped nursing every few seconds to catch her breath. On physical examination a loud, blowing systolic murmur was heard over the entire precordium. During the first nine weeks the infant gained only 2¼ pounds (1,021 Gm.), while the twin gained 4⅓ pounds (1,966 Gm.), and subsequently lost weight. Spells of rapid breathing were first noted when the infant was 11 weeks old. These increased in frequency and severity and were associated with cyanotic attacks. An infection of the upper respiratory tract developed during the thirteenth week, and several days later the child had a severe, prolonged cyanotic attack. She was admitted to the hospital at the age of 3 months and 11 days. Physical examination showed a rapid, regular heart rate and a loud systolic murmur transmitted to the base of the heart and the left axilla. Percussion showed enlargement of the heart, with dulness extending to the axillary line. The blood count showed 3,940,000 erythrocytes, 93 per cent hemoglobin, and 16,000 leukocytes, with 45 per cent polymorphonuclears and 55 per cent lymphocytes. The infant was placed in an oxygen tent, without benefit. She died six hours after admission. The older twin showed no sign of congenital defects during the year that she was seen in the outpatient clinic. Postmortem roentgen examination of the chest showed a greatly enlarged heart (fig. 6 *A*).

A complete autopsy was performed twenty hours post mortem. It showed a well developed, well nourished white girl, measuring 59.5 cm. in length. The diaphragm was at the level of the sixth rib on the right and of the sixth interspace on the left. There was a small amount of pleural effusion.

The heart weighed 84 Gm. (about twice the normal weight). It was symmetrically enlarged, and the pericardium was glistening. The total length of the heart was 90 mm., and the diameter at the base was 50 mm. The vessels entering the dilated and hypertrophied right auricle were in normal relation. The eustachian and thebesian valves formed a continuous membrane, the greatest width being 12 mm. (fig. 6 *B*). An anatomic and functional opening in the auricular septum, measuring 13 by 17 mm., was formed in part by a defect of the left superior portion of the septum primum and in part by a defect of the septum secundum along the inferior portion of the limbus ovalis. The wall of the hypertrophied right ventricle measured 7 mm. in thickness. There were two defects of the ventricular septum. The smaller defect, measuring 4 mm. in diameter, was located at the base, between the anterior and the medial cusp of the tricuspid valve, and its superior border, in the left ventricle, was located 4 mm. below the mid-

9. Shain, J. H.: Multiple Congenital Rib and Spinal Deformities: Report of a Case, J. Pediat. **3**:870 (Dec.) 1933.

point of attachment of the posterior cusp of the aortic valve. Its orifice, in the right ventricle, was partially covered by the high location of a short papillary muscle. The larger defect, measuring 13 by 7 mm., was located close to the right border of the septum, at a point one-third the distance from the base to the apex, below the junction of the medial and posterior cusps of the tricuspid valve. Its borders were smooth, thick and muscular, and five thickened chordae tendineae from the medial cusp of the valve were inserted in its medial inferior margin. The tricuspid valve showed normal anterior and posterior cusps, measuring from 8 to 10 mm. in length. The right half of the medial cusp measured 9 mm. in

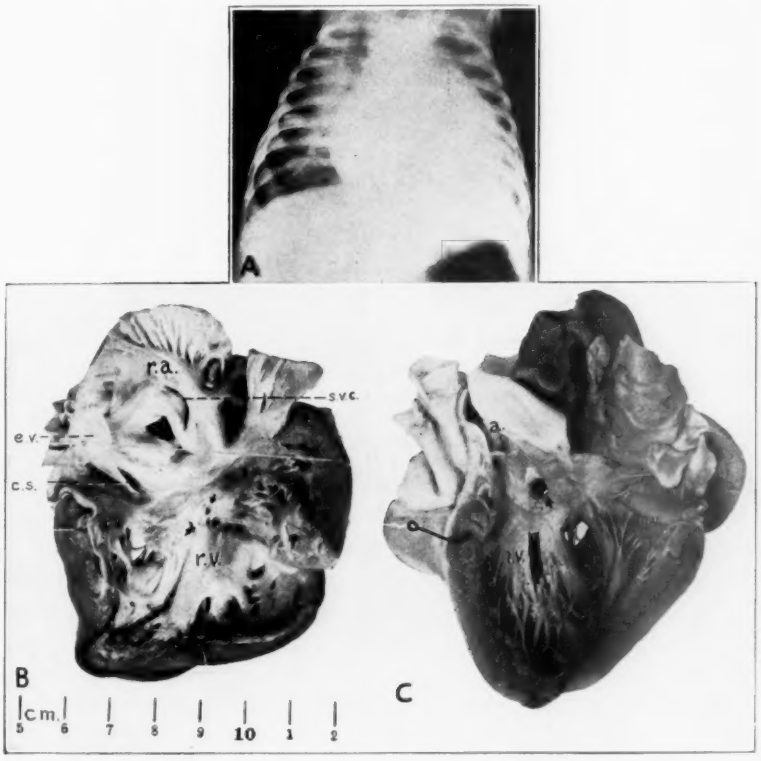

Fig. 6 (case 5).—*A*, postmortem roentgenogram of the chest; *B*, the right side of the heart. Three arrows point to the short, high papillary muscle, which lies in front of the smaller septal defect. *C*, the left side of the heart. The arrow points to the smaller septal defect, through which can be seen a papillary muscle. *a.* indicates aorta; *c.s.*, orifice of coronary sinus; *e.v.*, eustachian valve; *l.v.*, left ventricle; *m.v.*, mitral valve; *r.a.*, right auricle; *r.v.*, right ventricle; *s.v.c.*, orifice of superior vena cava; *t.v.*, tricuspid valve.

length, and its margins were thickened, glistening and somewhat nodular. The left half of this cusp measured only 4 mm. in length, and its papillary muscles and chordae were much shortened.

The pulmonary conus was normal. The circumference of the pulmonary valve was 40 mm., and its cusps were of equal size, thickened, translucent and glistening.

The pulmonary artery and its branches, together with the sinuses of Valsalva, were much dilated. The ductus arteriosus measured 5 mm. in diameter. The left side of the heart was normal throughout except for nodular thickening of the margins of the mitral valve and the septal defects, which are shown in the illustrations (fig. 6 *B* and *C*). Microscopic examination of sections from the cusps showed no fibrosis or evidence of inflammation. The aortic valve measured 46 mm. in circumference. The sinuses of Valsalva, the ascending aorta and the arch were much dilated. The coronary arteries were normal in origin and distribution. The thickness of the wall of the left ventricle was 9 mm. The circumference of the tricuspid valve was 56 mm.; of the mitral valve, 54 mm.; of the ascending aorta, 50 mm.; of the aortic arch, 30 mm., and of the thoracic aorta, 20 mm.

The left lung weighed 45 Gm. and the right 95 Gm. The left lung showed congestion at the base. The upper and lower lobes of the right lung showed dark patches of consolidation, revealed microscopically as areas of polymorphonuclear infiltration with foci of necrosis.

No variations of note were present in the other organs. The liver weighed 200 Gm. and the spleen 11.5 Gm. The brain was normal.

Cultures made at autopsy of blood from the right lung and heart showed a pure growth of Staphylococcus aureus.

The anatomic diagnosis included pneumonia of the right lung, with abscess formation, and the following congenital cardiac abnormalities: a double defect of the interventricular septum, a shortened medial cusp of the tricuspid valve, a defect of the auricular septum, patency of the ductus arteriosus, persistence of the common eustachian and thebesian valves and persistence of the embryonal thickening of the cusps of the pulmonary, mitral and tricuspid valves.

Comment.—The location of the defects of the ventricular septum in this case, involving, as they did, the papillary muscles of the medial cusp of the tricuspid valve, had caused a shortening of the medial cusp, as in the cases reported by Gutzeit [10] and Weinstein.[11] The position of the larger septal defect was unusual. The defect of the auricular septum represented a developmental arrest of the septum primum together with a defect in the fusion of the septum secundum with the valve of the left sinus in the formation of the limbus ovalis. The anomaly of the eustachian and thebesian valves was due to a lack of fusion of the inferior sinus septum and the right venous valve. Yater [7] noted that this anomaly was not uncommon in otherwise normal hearts.

COMMENTS ON THE CLINICAL FEATURES

The most important symptom in all the cases presented was cyanosis. It was least marked in the case in which there was a double septal defect, in which it did not occur until the eleventh week of life and

10. Gutzeit, K.: Ein Beitrag zur Frage der Herzmissbildungen an Hand eines Falles von kongenitaler Defektbildung im häutigen Ventrikelseptum und von gleichzeitigem Defekt in dem diesem Septumdefekt anliegenden Klappenzipfel der Valvula tricuspidalis, Virchows Arch. f. path. Anat. **237**:355, 1922.

11. Weinstein, S.: A Congenital Perforate Interventricular Septum of the Heart Accompanied by a Shortened Medial Tricuspid Leaflet and a Dilated Pulmonary Artery with Two Cusps, Tr. Chicago Path. Soc. **12**:279, 1926.

then was noted only occasionally. It was most marked in the case of transposition of the arterial trunks. In this case cyanosis became evident at birth; in the others it became evident some time after birth. The cyanosis was aggravated by exertion. Dyspnea or rapid breathing was not a prominent feature, although it was noted in two of the cases. Neither the dyspnea nor the cyanosis was relieved by placing the infant in an oxygen tent. The next important symptom was poor feeding. The infants did not take food well and did not gain well. A murmur was heard in all the infants that had a defect of the interventricular septum. It was systolic and loudest to the left of the sternum. Like the cyanosis, the murmur did not become evident until some time after birth; in one case, in which the infant was followed in the hospital from birth to death, a murmur was not heard until one month after birth. Polycythemia was present in only one case and was not marked (erythrocytes, 5,900,000; hemoglobin, 115 per cent). In the other cases there was, on the contrary, mild anemia. Although a diagnosis of congenital cardiac anomaly was made in all cases, no attempt was made to diagnose the type of anomaly present.

Dr. Louis C. Schroeder, director of the Pediatric Service of the New York Nursery and Child's Hospital at the time these infants were being observed, gave permission for the publication of these case reports.

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Case Reports

ESSENTIAL XANTHOMATOSIS

TREATMENT WITH CHOLESTEROL-FREE DIET IN TWO CASES

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Rowland [1] defined essential xanthomatosis as "a heredofamilial, constitutional disorder of metabolism in which a deposition of fat-like mixtures, particularly cholesterol and its ester, takes place in the tissues and blood. . . . The pathologic change appears in the form of granuloma-like accumulations arising in various parts of the body." Since an accumulation of excessive amounts of cholesterol is a dominant feature of this disease, restriction of cholesterol in the diet is a rational therapeutic procedure, and it has been tried in a few instances, with considerable success. Herrmann and Nathan [2] observed definite clinical improvement in two patients after treatment with a low cholesterol diet (*Pflanzenkost*), the description of which was not given. In one of them the cholesterol content of the blood serum decreased from 580 to 300 mg. per hundred cubic centimeters during four months on the diet; in the other the amount of serum cholesterol remained within normal limits throughout the period of study. Gaál [3] reported on similar treatment in a case of severe xanthomatosis, in which there were rather dramatic results. With a low cholesterol diet the amount of serum cholesterol fell from 660 to 170 mg. per hundred cubic centimeters in nineteen days. Simultaneously there was considerable clinical improvement. Schoenheimer [4] described extensive metabolic studies in a case

This investigation was assisted by a grant from the Josiah Macy Jr. Foundation.

From the Chemical Laboratory, Babies Hospital, and the Pediatric Service, Mount Sinai Hospital.

1. Rowland, R. S.: Anomalies of Lipid Metabolism, in Christian, H. A., and Mackenzie, J.: Oxford Medicine, London, Oxford University Press, 1932, vol. 4, pt. 1, p. 214.

2. Herrmann, F., and Nathan, E.: Zur Frage der Xanthomgenese, Arch. f. Dermat. u. Syph. **152**:575, 1926.

3. Gaál, A. M.: Untersuchungen über Cholesterinstoffwechsel bei Xanthomatosis essentialis, Ztschr. f. klin. Med. **113**:349, 1930.

4. Schoenheimer, R.: Ueber eine Störung der Cholesterin-Ausscheidung, Ztschr. f. klin. Med. **123**:749, 1933.

of essential xanthomatosis. He demonstrated that the accumulation of cholesterol in the tissues and blood was due to the absence of any excretion of cholesterol or its derivatives. When the patient was placed on a diet of food from plant sources, containing no cholesterol, the cholesterol content of the serum fell from an initial value of 850 mg. to 300 mg. per hundred cubic centimeters in fifty days, with some clinical improvement. Schoenheimer calculated that about 20 Gm. of cholesterol disappeared from the blood during this period; since it was not excreted it must have been destroyed. Apparently this patient had to rely entirely on a relatively slow process of degradation to eliminate excess cholesterol. When cholesterol was supplied continuously in the diet this process could not keep up with the demand, and cholesterol accumulated. Thannhauser [5] treated another patient with the same diet employed by Schoenheimer and obtained essentially the same result. In twenty-eight days the cholesterol content of the serum fell from 576 to 132 mg. per hundred cubic centimeters. In Thannhauser's case, as in Schoenheimer's, there was practically no excretion of cholesterol or its derivatives.

In the present report we shall describe a case of essential xanthomatosis with some unusual features, occurring in a child, and emphasize our inability to reduce the cholesterol content of the blood serum or to bring about any definite clinical improvement by long continued feeding of a cholesterol-free diet. The case resembled closely, both clinically and in the reaction to a cholesterol-free diet, another case, that of an adult, which is described briefly for comparison.

REPORT OF CASES

CASE 1.—*History.*—H. R., an 11 year old Jewish boy, was admitted to the pediatric service of Mount Sinai Hospital on April 17, 1934.

The mother, aged 31 years; the father, aged 43 years, and two brothers, aged 9 and 5 years, respectively, were well. There had been no miscarriages. There was no history of a condition similar to that exhibited by the patient on either the paternal or the maternal side of the family. There was no history of tuberculosis, heart disease, diabetes or cancer in the family.

The patient's birth was normal and occurred at term. Until he was 6 months old he cried continuously with colic, but he presented no feeding problem until he was 2 years of age, when he stopped eating well. There was severe diarrhea at that time, and there was a history of occasional diarrhea since infancy. He never had taken cod liver oil, but he received the juice of one orange daily after the third month. He had measles when he was 6 years old and chickenpox when he was 6½ years old, both without complications. Otherwise the past history was irrelevant except for features concerned with the present illness.

Present Illness.—At the age of 3 years blood was taken for examination during an infection of the upper respiratory tract, and it was noted that the serum was lipemic, so cream was removed from the diet for several weeks. When he was 4 years old about half a dozen xanthomatous areas, each approximately 0.5 cm. in

5. Thannhauser, S. J.: Ueber Lipoidosen, Klin. Wchnschr. **13**:161, 1934.

diameter, were observed in the deltoid region of the left arm. Despite treatment by a dermatologist similar areas appeared on the deltoid region of the right arm and on both elbows, the hips, the knees and the back. The patient was placed on a diet containing no cream, butter, carrots or fat meat, and except for occasional lapses this regimen has been followed to the present time. No eggs have been eaten during the past two years, and cheese and milk have been eliminated from the diet during the past four months. The following is a typical menu: breakfast—cereal (without cream or milk), tomato sandwich and Postum with 2 teaspoonfuls of skimmed milk; lunch—vegetable salad, fruit, bread, pie made with vegetable fat and Postum; dinner—a cooked vegetable (such as string-beans), baked beans without fat and boiled potatoes.

The changes in the height and weight since June 28, 1932 (when the patient came under the care of one of us [B. S.]) are shown in table 1. The loss of weight during 1934 may have been due to some restrictions in the diet or to thyroid medication, which was started late in December 1933 because of the finding of a somewhat low basal metabolism (1,050 calories, or a rate of —7 per cent) by Dr. Anne Topper.

TABLE 1.—*Changes in Height and Weight of H. R.*

Date	Age	Height, Cm.	Sitting Height, Cm.	Weight, Kg.
6/28/32	9 yrs., 6 wks.	136	73	33.0
12/23/33	10 yrs., 7 mos.	...	..	38.8
2/16/34	10 yrs., 9 mos.	143 (?)	73	40.8
4/ 4/34	10 yrs., 11 mos.	...	..	37.8
7/ 2/34	11 yrs., 2 mos.	144	75	37.6
12/22/34	11 yrs., 7 mos.	143	75	35.2
3/ 2/35	11 yrs., 10 mos.	145	76	36.2

During the past two years the patient had been treated with roentgen therapy, local implantation of radon seeds and a spray of carbon dioxide snow, with no definite effect.

Physical Examination.—Examination at the time of admission revealed a well developed, well nourished and cooperative 11 year old white boy in no distress and not acutely or chronically ill. Chiefly over the extensor surfaces but also over the flexor surfaces, at the joints of the elbows, knees and phalanges were irregular, elevated, sharply demarcated, yellowish wartlike plaques. Above the right elbow on the extensor surface some fine scarring had replaced the yellow plaques. Over the left elbow a yellow plaque was discolored with grayish spots. Small yellowish deposits were also present at the inner canthus of each eye (xanthelasma palpebrarum) and over the posterior portion of both external ears. The yellow, irregular plaques were most numerous over the buttocks and ilia. Near the anus some of the plaques were softer and some were pendulous. Over both shoulders and involving the anterior and posterior portion of the neck were irregular, yellowish macular areas, some of which were slightly elevated. The metacarpophalangeal joints of the fingers were thickened and somewhat deformed by the yellowish, firm deposits, as were the proximal interphalangeal joints and the distal interphalangeal joints of the fourth and fifth fingers of both hands. Above both heels and apparently involving the achilles tendon were soft deposits of tissue which appeared almost pendulous. There was a large ecchymotic area

over the anterior portion of the right tibia. The extent and nature of the lesions are best shown by the photographs.

There was a pronounced arcus juvenalis in the cornea of each eye, particularly at the upper and lower portions of the limbus. The fundi showed engorgement of the veins, particularly on the left side, and the arteries were moderately full and tortuous.

Otherwise no abnormalities of importance were noted. There were none of the characteristic anomalies of the diseases which are usually grouped [1] with essential xanthomatosis as disturbances of the lipid metabolism (Niemann-Pick's

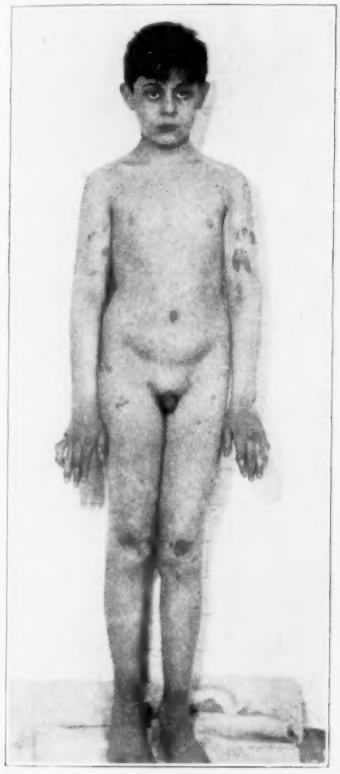

Figure 1

Figure 2

Fig. 1.—Distribution of plaques of xanthomatosis on the body.

Fig. 2.—Distribution of the plaques on the buttocks and ilia and on the left arm.

disease, Schüller-Christian's disease and Gaucher's disease). There were no soft areas in the skull or other lesions of the skeleton. The tip of the spleen was just palpable, and the liver was 2 cm. below the costal margin.

Course in Hospital and Treatment.—The patient was admitted primarily for study of the effect of a cholesterol-free diet on the cholesterol metabolism and general condition. He was given the usual ward diet for the first sixteen days and then placed on a diet containing only foods of plant origin. A sample diet

Fig. 3.—Lesions on the hands, causing deformity and thickening of the joints of the fingers.

Fig. 4.—Soft, almost pendulous deposits on the heels and ecchymotic area on the anterior portion of the right tibia.

is shown in table 2. Since his discharge from the hospital the patient has continued on essentially the same diet, except for the addition of 500 cc. of skimmed milk daily.

The cholesterol (total, free and combined [estimated as the difference between the total amount of cholesterol and the amount of free cholesterol]) content of the blood serum was determined at frequent intervals by the method of Schoenheimer and Sperry [6] both during and since the patient's stay in the hospital. The results, including several analyses of serum from the mother, father and brothers, are set forth in table 3. Omission of cholesterol from the diet had no measureable effect on the cholesterol content of the serum. There were some wide fluctuations in the total cholesterol content of the serum, but no significant decrease from the original high level occurred. The ratio of combined cholesterol to free cholesterol remained constant, within the limit of error of the analysis, throughout the period of study.

During the patient's stay in the hospital there was no change in the clinical symptoms. Since his discharge his appearance has remained essentially the same, though his mother has the impression that some of the yellow areas are less

Fig. 5.—Arcus juvenalis in the cornea.

elevated, that the skin in the center of the areas over the knee joints is more nearly normal in appearance and that there is more mobility in the fingers.

Thyroid medication (tablets containing 0.015 Gm., given three times daily) was continued during the patient's stay in the hospital and until January 1935, when it was stopped. Since the hypercholesteremia has persisted for at least five months after the withdrawal of thyroid, it is hardly likely that the failure of the cholesterol-free diet to reduce the amount of cholesterol in the serum was due to the administration of thyroid. Determinations of the basal metabolism while the patient was in the hospital gave the following values: On April 19, 1934, a test of the basal metabolism showed 1,239 calories, or a rate of +15 per cent; on May 24 the number of basal calories was 1,176 (a rate of +7 per cent), and on June 3 the number of basal calories was 1,123 (a rate of +3 per cent).

Frequent examination of the blood and the urine revealed no abnormalities except the hypercholesteremia.

6. Schoenheimer, R., and Sperry, W. M.: A Micromethod for the Determination of Free and Combined Cholesterol, J. Biol. Chem. **106**:745, 1934.

TABLE 2.—*Sample Diet of Foods from Plant Sources*

Constituent	Amount, Gm. or Cc.	Carbohydrate, Gm.	Protein, Gm.	Fat, Gm.
Breakfast				
Grapefruit juice	150	13	1	..
Orange	150	18	1	..
Sugar	34	34	..	..
Bread	50	26	4	..
Honey	20	20	..	..
		111	6	
Dinner				
Asparagus	100	3	2	..
Lettuce	100	3	2	..
Apple-sauce (sweet)	100	40	1	..
Bread	50	26	4	..
Honey	20	20	..	..
Orange juice	200	18	2	..
Sugar	17	17	..	..
Vegetable oil	10	..	..	10
		127	11	10
Supper				
Cole slaw with 10 Gm. of vegetable oil	100	3	2	10
Sweetened apricots	100	20	1	..
Bread	50	26	4	..
Grapefruit juice	200	18	2	..
Sugar	34	34	..	..
		101	9	10
Total		339	26	20
Calories, 1,640				

TABLE 3.—*Cholesterol Content of Blood Serum of H. R. and of His Parents and Brothers*

Subject	Date	Cholesterol, Mg. per 100 Cc.			Ratio of Combined Cholesterol to Free Cholesterol
		Total	Free	Combined	
H. R.	4/17/34	Admitted to hospital			
	4/18/34	810	224	586	2.62
	4/21/34	869	238	631	2.65
	4/24/34	685	202	483	2.39
	4/27/34	845	235	610	2.60
	4/30/34	870	255	615	2.62
	5/ 3/34	948	264	684	2.59
	5/ 4/34	Special diet of food from plant sources started			
	5/ 5/34	909	259	650	2.51
	5/ 8/34	955	281	674	2.40
	5/11/34	767	224	543	2.42
	5/14/34	817	235	582	2.48
	5/18/34	833	238	595	2.50
	5/21/34	926	265	661	2.49
	5/25/34	912	275	637	2.32
	5/31/34	810	235	575	2.45
	6/ 4/34	Discharged from hospital			
	6/18/34	765	224	541	2.42
	6/22/34	815	241	574	2.38
	7/ 3/34	795	227	568	2.50
	7/10/34	772	226	546	2.42
	10/ 2/34	853	247	606	2.45
	5/18/35	762	209	553	2.65
Mother	6/ 4/34	478	125	353	2.82
	10/ 2/34	410	110	300	2.73
	5/18/35	427	114	313	2.75
Father	6/18/34	419	119	300	2.52
D. R., aged 9 yrs. (brother)	6/ 4/34	390	102	288	2.82
H. R., aged 5 yrs. (brother)	6/ 4/34	160	41	119	2.90

Roentgen examination of the legs on May 16, 1934, showed no evidence of calcification of vessels.

CASE 2.—*History.*—J. J., a white man 30 years old, was first admitted to the Presbyterian Hospital on Oct. 26, 1932, with a long history of rheumatic attacks and "lumps" on the fingers, elbows, knees, heels and buttocks.

Both parents were living and well. There was no history of any condition in any member of the family similar to that presented by the patient. The past history was irrelevant.

Present Illness.—The first "rheumatic attack" occurred on Christmas day, 1921, and lasted about four days. The only symptom was considerable pain in the heels. Similar attacks, in most of which only the knees were involved, have occurred at frequent intervals (about four times a year) since. Shortly after the first attack a "lump" was noted on the middle finger of the right hand. This grew slowly for four years and was excised but grew again. About 1926 another lump developed on the fourth finger of the left hand; it was excised in 1931. The diagnosis of xanthoma tuberosum multiplex was made at the New York Post-Graduate Medical School and Hospital on the basis of examination of excised nodules.

During the past four years lumps have grown on both elbows, and on the knees, the heels and the buttocks. Yellow patches have been present on the eyelids for at least nine years. There is no history of glycosuria. The patient does not like vegetables and, until three years ago, ate a diet consisting largely of meat with little carbohydrate. During the past six months he has received a high carbohydrate, low fat diet plus insulin, with no apparent effect on the lesions.

Physical Examination.—The results of physical examination were irrelevant except for the following points: There were flat yellow nodules over both wings of the scapulae and also above the clavicles. There was a sore, yellowish-red polypoid lump, 3 cm. in length, at the base of the spine. There were many nodules, some hard and attached to bone, others soft and movable, some yellow and others covered with normal skin, over the olecrana, calcanea, tibias, fibulas, achilles tendons, metatarsophalangeal joints of the great toe and especially on the fingers, as was noted in the preceding section. There were large xanthomas on the right upper and left upper and lower eyelids. There was a marked arcus corneae.

Course in Hospital and Treatment.—After a preliminary period of nine days on a constant diet containing a moderate amount of animal products the patient was placed on a diet consisting entirely of plant products except for 500 cc. of skimmed milk per day. The skimmed milk was shown to contain negligible amounts of cholesterol.

The cholesterol content of the blood plasma[7] was determined at frequent intervals by a macromodification[8] of the method of Schoenheimer and Sperry.[6]

7. The plasma was oxalated. Recently it has been found (Sperry, W. M., and Schoenheimer, R.: A Comparison of Serum, Heparinized Plasma, and Oxalated Plasma in Regard to Cholesterol Content, J. Biol. Chem. **110**:655 [Aug.] 1935) that oxalated plasma contains significantly smaller amounts of cholesterol than either serum or heparinized plasma. Hence the values shown are undoubtedly lower than would have been found in serum.

8. Sperry, W. M., and Stoyanoff, V. A.: Effects of Long-Continued Cholesterol Feeding in Rats, J. Nutrition **9**:145, 1935.

The results (table 4) show no marked effect of the cholesterol-free diet. The amount of total cholesterol was not excessively high before the diet of foods from plant sources was started. There was an apparent decrease from over 400 to a little over 300 mg. per hundred cubic centimeters, but little significance can be attached to this in view of the wide fluctuation, with a decrease to as low as 332 mg., before the diet was instituted. Furthermore, the patient, who was highly intelligent and, we believe, trustworthy, and who understood thoroughly the purpose of the diet, claimed that he had adhered to it rigorously after returning home. Despite this the total cholesterol content was over 400 mg. per hundred cubic centimeters two months after his discharge from the hospital.

There was no change in the patient's clinical condition during his stay in the hospital. Two months after his discharge it was found that three new masses had developed and that other masses had increased in size despite continuance of the cholesterol-free diet.

TABLE 4.—*Cholesterol Content of Blood Plasma of J. J.*

Date	Cholesterol, Mg. per 100 Cc.			Ratio of Combined Cholesterol to Free Cholesterol
	Total	Free	Combined	
10/28/32	447	137	310	2.26
11/ 2/32	332	...	...	....
11/ 7/32	408	119	289	2.43
11/ 8/32	Cholesterol-free diet started			
11/10/32	456	113	343	3.04
11/15/32	368	106	262	2.47
11/19/32	436	...	...	....
11/22/32	323	91	232	2.55
11/25/32	300	91	209	2.30
11/29/32	335	101	234	2.32
12/ 3/32	308	83	225	2.71
12/ 4/32 Discharged from hospital				
12/12/32	342	98	244	2.49
1/ 9/33	366	103	263	2.55
2/ 6/33	410	118	292	2.47
3/ 6/33	382	116	266	2.29

While the patient was in the hospital xanthomatous masses were excised from the middle finger of the right hand by Dr. Hugh Auchincloss. A sample contained 4.3 per cent of cholesterol (20 per cent on the basis of dry weight), of which 12 per cent was free and 88 per cent was combined.

COMMENT

While essential xanthomatosis can hardly be regarded as a rare disease, the first case which we have described was of particular interest because the clinical picture resembled closely that usually seen only in long standing cases of the disease in adults. This resemblance can be brought out best by comparison with the typical case in an adult (case 2), which one of us (W. M. S.) had the opportunity to observe through the courtesy of Dr. Alvin F. Coburn. One feature of both cases was xanthelasma palpebrarum, which, although not unknown in childhood, is seldom seen at that age. It is the most characteristic manifestation

of the disease in adults and, according to Rowland,[1] probably represents "a slowly developing, long standing form of xanthomatosis."

Another outstanding aspect of both cases was the existence of a marked arcus corneae, which, although it has been noted in the description of a few cases, has not been emphasized as characteristic of essential xanthomatosis. Rowland did not mention it in his comprehensive description of the disease. It is of interest in this connection that an arcus of the cornea develops in cholesterol-fed rabbits simultaneously with atherosclerosis and deposits of cholesterol in organs.

Both cases were characterized by deep-seated lesions at the joints, especially the joints of the fingers, and at the achilles tendons. Such lesions are considered to represent a process of long standing, and, though they are not unknown in children, are regarded as more characteristic of the disease in adults. The cutaneous lesions, which were present in abundance, especially over the shoulders and neck, in H. R., and which were absent in J. J., are thought to represent an early stage of the disease.

In neither case were there any complications except the "rheumatic attacks" in J. J., which may or may not have been related to the xanthomatosis. In both cases the disease was "essential" as defined by Rowland.[1]

The finding of hypercholesteremia in the mother, father and one brother of H. R. emphasizes the heredofamilial nature of essential xanthomatosis and is of particular interest since no surface lesions have developed in them. If one assumes that the disease is present in latent form, the fact that an increased cholesterol content of the blood serum is the only apparent abnormality of the parents and brother is of interest in connection with the hypothesis that hypercholesteremia is the primary disturbance in the disease. This hypothesis has been widely accepted, mainly because the amount of cholesterol in the serum has been shown to be abnormally high in most cases of essential xanthomatosis. However, the finding of some cases in which there was no increase in the amount of serum cholesterol has been difficult to explain and has led Bloch[9] to suggest that the deposition of cholesterol and other fatty substances in cases of xanthoma is due to abnormalities in the proportionate amounts of the various lipids in the blood serum. He has emphasized particularly variations from the normal ratio of combined cholesterol to free cholesterol as being of primary importance in the etiology of the disease. In both H. R. and J. J. the ratio was normal throughout the period of study.

The administration of a cholesterol-free diet over a long period of time had little effect on the amount of serum cholesterol or on the

9. Bloch, B.: Metabolism, Endocrine Glands and Skin Diseases, with Special Reference to Acne Vulgaris and Xanthoma, Brit. J. Dermat. **43**:61, 1931.

clinical condition in either H. R. or J. J. It seems probable, therefore, that the primary cause of the disturbance in our cases was not a failure in excretion, since Schoenheimer [4] and Thannhauser [5] obtained a marked effect with similar treatment of their patients, in whom the condition was caused by inability to excrete cholesterol. Unfortunately, we have not been able to determine the amount of cholesterol excreted, as was done by these authors, but in the case of J. J. we measured the intake and output of unsaponifiable material (containing the sterols) during one period of five days while the patient was on a normal mixed diet and during five periods of five days while he was on the cholesterol-free diet. During the control period there was a positive balance of 6 Gm., but during the periods when the patient was on the cholesterol-free diet there was an average negative balance of 2 Gm. per five-day period, indicating an excretion of excess sterol. However, this finding is open to question, since it is difficult to extract sterols quantitatively from plants. Even though samples of the food were treated exhaustively for many days with boiling alcohol, it is still possible that the negative balances were due to incomplete extraction of the sterols.

An outstanding difference between the cases described by Schoenheimer and Thannhauser and our cases is in the ratio of combined cholesterol to free cholesterol of the blood serum. One of us [10] has found, in disagreement with most previous investigators, that this ratio is quite constant in normal persons. In one hundred and twenty-six samples of serum from ninety-one healthy adult human subjects a minimum ratio of 2.31 and a maximum ratio of 3.12 were obtained, which is to say that the proportion of combined cholesterol in the total cholesterol of the serum varied between approximately 70 and 75 per cent only. It will be noted that in both our cases most of the values for the ratio of combined cholesterol to free cholesterol fell within the normal range. By contrast, in Schoenheimer's case the ratio was 16; i. e., 94 per cent of the total cholesterol content of the serum was in combined form. In several hundred determinations of the amount of cholesterol in serums from patients with various pathologic conditions we have never found a ratio of this magnitude; in a negligible number the ratio was above the normal range, 4 (80 per cent of the total amount of cholesterol in combined form) being the maximum. A ratio such as that observed by Schoenheimer appears to be exceedingly rare. In Thannhauser's case the ratio varied greatly from the normal in the opposite direction, being 0.17 (15 per cent of the total amount of cholesterol in combined form). The greatly decreased proportion of combined cholesterol was probably associated with the presence of cirrhosis

10. Sperry, W. M.: The Relationship Between Total and Free Cholesterol in Human Blood Serum, J. Biol. Chem. **114**:125 (May) 1936.

of the liver in this patient, since it has been pointed out by Thannhauser and Schaber [11] and confirmed by Epstein [12] that such low ratios are associated with hepatic disease.

The marked difference between our patients and those of Schoenheimer [13] and Thannhauser in regard to the distribution of cholesterol in the serum indicates that our failure to obtain the effects observed by these authors with a cholesterol-free diet was due to some fundamental difference in the pathogenesis of the disease. The primary purpose of this paper is to point out that such differences exist.

In a recent study one of us [14] demonstrated that a considerable esterification (average, 57 per cent) of the free cholesterol of normal blood serum occurs on incubation. Serum from both H. R. and his mother has been found to react in the same way. On incubation for three days 25 per cent of the free cholesterol in H. R.'s serum and 38 per cent of the free cholesterol in the mother's serum was esterified. Apparently these serums were normal in regard to cholesterol esterase activity. The degree of esterification in the case of H. R. was slightly less than the minimum (29 per cent) which has been found previously, but we attach no significance to this, since there is a wide variation in the degree of reaction of normal blood serum.

SUMMARY

A case of essential xanthomatosis occurring in a child is described. The case is of interest because of certain features, particularly the presence of xanthelasma palpebrarum and a marked arcus corneae, which are more characteristic of the advanced form of the disease as it occurs in adults. A description of such an advanced case in an adult is included for comparison.

Treatment for considerable periods of time with a cholesterol-free diet had no definite effect, either on the hypercholesteremia or on the clinical picture of either of the patients. The results are in contrast to those obtained by other investigators, who have reported a marked decrease in the cholesterol content of the blood serum and an improvement in the clinical condition following the administration of similar cholesterol-free diets to patients with essential xanthomatosis. There appear to be marked differences in the pathogenesis of this disease,

11. Thannhauser, S. J., and Schaber, H.: Ueber die Beziehungen des Gleichwichtes Cholesterin und Cholesterinester im Blut und Serum zur Leberfunktion, Klin. Wchnschr. **5**:252, 1926.

12. Epstein, E. Z.: The Cholesterol Partition of the Blood Plasma in Parenchymatous Diseases of the Liver, Arch. Int. Med. **47**:82 (Jan.) 1931.

13. The study of H. R. was carried out with the advice of Dr. R. Schoenheimer, who is in complete accord with the opinions expressed in this paper.

14. Sperry, W. M.: Cholesterase in Blood, J. Biol. Chem. **111**:467, 1935.

since some patients react favorably to a cholesterol-free diet while others are not affected by the same treatment. All patients with essential xanthomatosis should be treated with a cholesterol-free diet for a considerable period of time, since a certain number may be expected to react favorably.

In our cases the proportion of combined cholesterol to free cholesterol in the serum was normal; in the previously described cases, in which the patients showed improvement on a cholesterol-free diet, the ratio of combined cholesterol to free cholesterol was highly abnormal. This may prove to be a point of diagnostic importance, and we suggest that the amounts of combined cholesterol and free cholesterol be determined before and during treatment with a cholesterol-free diet in all cases of essential xanthomatosis.

ABSCESS OF THE LIVER OF UMBILICAL ORIGIN IN INFANTS

REPORT OF TWO CASES

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Abscess of the liver in infants is rare and is usually not taken into consideration in the differential diagnosis of obscure lesions in the upper half of the abdominal cavity. In this article 2 cases of abscess of the liver in infants who came to autopsy will be reported, and a discussion of the pathogenesis in relation to umbilical infection will be given.[1]

REPORT OF CASES

CASE 1.—A white boy 6 weeks of age was brought to the hospital by the mother because of swelling and tenderness of the abdomen and because of regurgitation of food, both of two weeks' duration. The infant was perfectly well until two weeks before admission, when the mother first noticed a small mass in the right upper abdominal quadrant. The mother continually rubbed and massaged the mass in the belief that it would thereby disappear. However, the mass continued to increase in size, and the consistency remained firm. The patient began to regurgitate his feedings about the same time that the mass was discovered. Regurgitation occurred from fifteen to twenty minutes after feeding, but only a small amount of vomitus was thrown up. The mother stated that the feedings oozed out from the mouth and that there was never any forceful spitting or vomiting. Tenderness was noticed over the mass, which appeared to have become progressively worse with the increase in size. Before admission a physician was called, who said that the baby had a temperature of 103 F.

The infant was the second child and had been born at full term by spontaneous delivery; the weight at birth was 9 pounds and 6 ounces (4,252.4 Gm.). He had been breast fed. The mother had had no miscarriages.

Examination revealed a fairly well nourished and fairly well developed white boy, who appeared ill and had a temperature of 99.8 F. but was not toxic. The head, ears, eyes, nose, throat, lungs and heart were essentially normal. The abdomen was distended and tender on palpation. A moderately hard mass was felt in the right upper abdominal quadrant in the hepatic region. No apparent rigidity was present. The extremities and genitalia were normal. The impression at that time was that there was a tumor of the kidney pressing on the gastrointestinal tract, producing vomiting.

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1. In preparing this article I referred freely to the work of the following: Cullen (The Umbilicus and Its Diseases, Philadelphia, W. B. Saunders Company, 1916, pp. 74-84), Maygrier (Bull. Soc. d'obst. de Paris **4**:146, 1901) and Leblond (Diagnostic et traitement des abcès du foie, Thèse de Paris, no. 1, 1892).

During his short stay in the hospital the patient slept and cried at intervals, regurgitating yellow-green fluid. The temperature varied from 99.8 to 99.2 F.; the respiratory rate, from 50 to 60. The infant became cyanotic and expired twelve hours and thirty minutes after admission.

Postmortem Observations.—The body was that of a well nourished white male infant. Yellowish material exuded from the nose. The mucous membranes of the mouth and lips were pale, and the finger-tips were cyanotic. The abdomen was distended and slightly above the level of the chest. There was an irregular mass in the right upper abdominal quadrant. The umbilical cicatrix appeared normal.

When the abdomen was opened pus escaped. The anterior surface of the right lobe of the liver was loosely attached to the posterior surface of the anterior abdominal wall by fibrous tags intermingled with pus. The liver was 50 and 30 mm. below the xiphoid arch and the costal arch, respectively. The abdominal cavity contained about 150 cc. of pus. The serosa of the intestines was slightly injected and in places covered with fibrin. The sigmoid colon was long, extended upward and was adherent to the lower border of the liver. The transverse colon, which was located below the sigmoid colon, was also attached to the inferior surface of the liver.

The liver measured 16 by 9 by 4.5 cm. The anterior surface of the right lobe showed a slightly depressed area 7 by 5.5 cm. in diameter. This area extended close to the falciform ligament. In this region the capsule of the liver was thickened, varied in color from dark purple-gray to red-gray and was shaggy. Under the capsular thickening of the right lobe there was an area measuring 30 by 25 by 40 mm., which was composed of numerous small pus-filled cavities; these averaged 2 mm. in diameter. These cavities were surrounded and separated by bright yellow-gray lines. The remaining portion of the liver was light purple-brown and moist and was without acinar markings.

The spleen weighed 45 Gm. It was covered by loosely adherent fibrinous membranes. The cut surface was purple-gray; the follicles were distinct. The other organs were essentially normal.

A smear from the abscess of the liver revealed many degenerated pus cells, with clumps of large gram-positive cocci. A smear from the abdominal cavity contained numerous pus cells, gram-positive cocci in diplococcic form and in clumps and a few gram-negative bacilli. Culture from the pus of the abscess disclosed many colonies of Staphylococcus citreus and a few colonies of Bacillus coli.

Microscopic Appearance of the Liver in the Region of the Abscess.—Glisson's capsule was markedly thickened and was vascular and infiltrated by round cells. It was most markedly vascularized on the inner side. The lobules of the liver were disrupted and hardly distinguishable except for remnants in the form of cords of cells. These cells had a slightly granular cytoplasm and in some places contained granules of pigment. There were large and small focal accumulations of polymorphonuclear leukocytes, which were fairly well preserved. Between these leukocytes a moderate number of mononuclear cells were noted. The connective tissue adjacent to the accumulations of pus cells was very cellular, and between fibrocytes there were small round cells and histiocytic elements.

Microscopic Appearance of the Abdominal Wall in the Region of the Abscess.—The muscle fibers were separated by an increased amount of connective tissue and perivascular accumulations of round cells and histiocytes. The subserosa was thickened and cellular, as was the deep fascia. On the inside of the wall was vascular granulation tissue, which gradually merged into a dense accumulation of polymorphonuclear leukocytes.

Microscopic Appearance of the Diaphragm.—The surface of the diaphragm, facing the abscess, was covered by cellular granulation tissue, which in turn passed into the covering coat of fibrin, mononuclear cells and leukocytes.

The anatomic diagnosis was: abscess in the right lobe of the liver, perihepatic abscess over the right lobe of the liver, diffuse suppurative peritonitis, displacement of the sigmoid colon to the lower border of the right lobe of the liver, marked cloudy swelling of the kidneys, anemia and cloudy swelling of the myocardium and petechial hemorrhages in both lungs.

Case 2.—A Mexican boy 6 weeks of age was brought to the Cook County Hospital by his mother. According to her, the infant was well until six days previously, when he began to spit up his feedings. A physician was then consulted, who prescribed medicine, following which the infant appeared to get along well for a day or two. On the third, fourth and fifth days of his illness the bowels did not move, but on the sixth day the bowels began to move again. On the fifth day of his illness he began to appear worse and vomited everything given to him.

The infant was the mother's third child. Birth had been by normal delivery. The weight at birth was 9 pounds (4,082.3 Gm.). The mother had had no miscarriages.

Examination revealed a well developed, well nourished Mexican boy, 1½ months old, weighing 10 pounds and 8 ounces (4,762.7 Gm.), with a temperature of 102 F. The fontanels were open. The only positive finding was a distended abdomen, which was tympanitic but not rigid. Because of resistance to palpation there appeared to be some tenderness in the right upper quadrant of the abdomen.

The day after admission, the temperature was still elevated (102 F.), but the patient took feedings well. On the third day in the hospital the infant appeared to be more irritable. On examination the abdomen was still distended. Impaired resonance was first noticed over the lower lobe of the left lung and the possibility of pneumonia was considered. Two days later the infant appeared like an afebrile child but still regurgitated part of the feedings. On the seventh day of his stay in the hospital he cried a great deal and was still regurgitating part of the feeding. The stool appeared yellow and soft. Examination of the chest revealed impairment of resonance on the right side also. The temperature rose to from 103.4 to 104 F. during this day. The next day the abdomen did not appear very distended, but a distinct mass was palpable about the umbilicus. The stool became watery and frequent. The temperature was high (from 103.2 to 105.6 F.). The ear drum appeared injected and was incised. Operative intervention was considered but was not done, owing to the condition of the patient, which became progressively worse. He died eleven days after admission to the hospital. The final diagnosis was bronchopneumonia with nutritional disturbance. The nature of the mass was still in doubt.

Postmortem Observations.—The body was much emaciated; the skin was pale and dirty gray, and the abdomen was distended. In the region of the umbilicus a firm fluctuating swelling about 5 cm. in diameter was felt.

In the region of the umbilicus, between the anterior abdominal wall and several loops of the lower part of the ileum was a well defined cavity 4 by 2.5 by 3 cm. in diameter, lined by a fibrinous membrane the inside of which was light brown and granular. The cavity contained about a tablespoonful of thick yellow-gray pus. Attached to the upper aspect of the cavity and fusing with its wall was the umbilical vein, which was greatly thickened. Its diameter was 5.5 mm. The wall of the vein was formed by dense fibrotic whitish tissue. The lumen was filled

by light gray and slightly transparent tissue, with deposits of a soft butter-like yellow material. In one place near the liver the lumen was patent, measured 2 mm. in diameter and was filled by yellow pus.

The liver weighed 130 Gm. It was moderately firm and pale brown-gray. In the lateral part of the convex surface of the right lobe was a subcapsular area 5 cm. in diameter, which was filled by light yellow-gray pus. The remaining portion of the liver was light brown-gray and was without acinar markings. The left lobe of the liver was distinctly darker brown than the right.

The intestinal serosa was slightly injected and was of peach blossom color, with linear injections parallel to the mesenteric attachment. The intestinal content was thin, and was light yellow-brown. The other observations were essentially negative.

Smears from the abdominal abscess and umbilical vein revealed numerous extracellular gram-positive staphylococci and many degenerated pus cells.

Microscopic Appearance.—The wall of the abscess, between the liver and the anterior abdominal wall, consisted of cellular granulation tissue rich in histiocytes and polymorphonuclear leukocytes, which passed into the wall of the vein without a line of demarcation. The inside was covered by shrunken and degenerated pus cells. Distal to the abscess the wall of the umbilical vein was thick, and the lumen was filled by a moderately vascular connective tissue. There were only a few small groups of pus cells in the outer layer of the wall. The abscess in the right lobe of the liver was located on the surface, the underlying parenchyma being undisturbed. Throughout the liver the Kupffer cells were swollen and mobilized, and many of them contained blood pigment.

The anatomic diagnosis was chronic suppurative thrombophlebitis of the umbilical vein, with formation of a large intra-abdominal abscess; a single large abscess on the surface of the right lobe of the liver; recent diffuse peritonitis; focal bronchopneumonia in the lower lobes of both lungs, and moderate emaciation.

EMBRYOLOGY AND ANATOMY OF THE UMBILICAL REGION

Before considering the pathogenesis of abscess of the liver in infants it is well to discuss briefly the anatomy and development of the umbilical region. The anterior abdominal wall begins to form in the 6 mm. embryo [2] both above and below the umbilicus. In a 10 mm. embryo the cord is well formed.

The anlage of the liver is first recognized in the 4.5 mm. embryo.[3] It grows downward and ventrally, coming in contact with the diaphragm and the anterior abdominal wall. It meets and surrounds the omphalomesenteric veins, which later form the portal vein. In addition to receiving blood from the intestines through the portal vein, the liver early receives blood from the placenta through the left umbilical vein. In the 6.5 mm. embryo the left umbilical vein has become the chief outlet for the placental blood.

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2. Politzer, V., and Sternberg, H.: Ztschr. f. Anat. u. Entwcklngsgesch. **92**:279, 1930.

3. The information concerning the development of the liver was taken from Keibel, F., and Mall, E. P.: Manual of Human Embryology, Philadelphia, J. B. Lippincott Company, 1912, vol. 2, pp. 291 and 406.

At birth the umbilical vein courses toward the liver, suspended by the falciform ligament. If the ligament is short the vein lies close to the anterior abdominal wall.[4] In many cases, however, the triangular falciform ligament between the umbilical vein and the anterior abdominal wall is wide and redundant and covers completely the right lobe of the liver.[4] At the umbilicus the peritoneum presents either a smooth surface or a slight depression; it may be adherent to the skin but can be easily separated.[5]

The lymphatics of the anterior abdominal wall may be divided into three groups [6]—the superficial cutaneous lymphatics, the deep cutaneous lymphatics and the lymphatics of the umbilical aponeurosis. The first group drain into the axillary and inguinal lymph nodes but anastomose, according to Cuneo and Marcelle,[6] with those about the umbilical vein and arteries. L'lorcia [7] stated that this system also anastomoses with the posterior intrathoracic lymph nodes. The lymphatics of the third group mentioned drain into the external iliac lymph nodes.

The set of lymphatics of most interest are those of the second group, the deep cutaneous lymphatics. These lymphatic channels from the umbilical region pass on either side of the falciform ligament, pierce the diaphragm and enter the anterior mediastinal lymph nodes. They communicate with the lymph channels about the umbilical vein which enter the liver.

The umbilicus itself is supposed to have no definite vessels but possibly has ill defined lymph channels in the matrix of Wharton's jelly. Scott [8] has claimed that he could trace the lymph channels along the umbilical vein for a distance of 4 cm. into the cord.

PATHOGENESIS OF UMBILICAL INFECTIONS WITH REGARD TO ABSCESS OF THE LIVER

In order to understand the umbilical infections the normal bacteriologic flora of the umbilical stump has to be considered first. Laurinsich [9] made 350 tests on the umbilical stump of the new-born, and came to the conclusion that 18 per cent of the babies had pathogenic micro-

4. Personal observations of the author.

5. Gauderon, quoted by Cullen, T. S.: The Umbilicus and Its Diseases, Philadelphia, W. B. Saunders Company, 1916, pp. 45 and 46.

6. Delamare, G.; Poirier, P., and Cuneo, B.: The Lymphatics, translated and edited by C. H. Leaf, London, Constable & Co., Ltd., 1913, p. 148.

7. L'lorcia, quoted by Rouvière, H.: Anatomie des lymphatiques de l'homme, Paris, Masson & Cie, 1932, pp. 294 and 295.

8. Scott, quoted by Minot, C. S.: Human Embryology, Baltimore, William Wood & Company 1892, p. 356.

9. Laurinsich, A.: Pediatria **32**:1022, 1924.

organisms. Adair [10] examined the umbilical stump in 65 cases and found pathogenic bacteria in 19.46 per cent. Küstner,[11] who examined the umbilical stump in 200 cases, encountered Staphylococcus albus-nonhaemolyticus in over 26.5 per cent and Bacillus coli-communis in 24.3 per cent. The other organisms present were Staphylococcus albus-haemolyticus, Streptococcus nonhaemolyticus, bacilli of the colon group, diphtheroid bacilli, the diphtheria bacillus and the hay bacillus.

Diphtheria organisms are occasionally present in the umbilical stump. Recently, Signy and Bruce [12] collected 30 instances of authentic umbilical diphtheria. The presence of the aforementioned organisms in from 19 to 23 per cent of the cases, even under aseptic conditions in a modern delivery room, is significant. The devitalized umbilical cord forms an excellent culture medium for bacteria, with the body supplying the optimum temperature for growth. If the umbilical cord is covered with thick gauze in such a way as to prevent evaporation, the organisms will have all the necessary requirements for their growth and multiplication. The bacterial growth in the umbilical stump becomes a serious event in the case of low resistance of the infant. Of particular importance in this regard is congenital syphilis, which often affects the umbilical stump in the form of syphilitic omphalitis associated with miliary gumma formation.[13]

Another factor that must be considered is the ultimate obliteration of the umbilical vessels at the level of the umbilicus after separation of the stump. The obliteration of the umbilical vessels, especially of the vein, the musculature of which is not as well developed as in the arteries, takes place very slowly, and a fine communication often persists between the lumen of the vein and the bottom of the umbilical hollow (and through it to the outside).[14] This is most frequent in cachetic premature infants in whom these unobliterated vessels persist and who are exposed to infection, with the subsequent development of phlebitis. The organisms can thus be disseminated through the umbilical vein, portal vein, umbilical arteries, lymphatics and structures about the umbilical vein and through arteries to the peritoneum, liver, mediastinum, inguinal region and other parts.

The umbilical wound is most frequently the point of entry of the infective material in the new-born. This was proved by Runge in 30 of 36 autopsies. The infective material is carried by direct contact.

10. Adair, F.: Care of the Umbilical Stump: A Bacteriologic Study, J. A. M. A. **41**:537 (Aug. 23) 1913.

11. Küstner, H.: Ztschr. f. Geburtsh. u. Gynäk. **84**:771, 1922.

12. Signy, A. G., and Bruce, A.: Arch. Dis. Childhood **7**:43, 1932.

13. Dominici, M.: Riv. di clin. pediat. **9**:613, 1911.

14. Audion, P.: Contribution à l'étude de l'ombilic et des infections ombilicales chez le nouveau-né, Thèse de Paris, no. 313 (April) 1900, p. 26.

The infection of the umbilicus often produces phlebitis of the umbilical vein (Porak and Durante [15]), which sends some branches into the parenchyma of the liver to anastomose with the intrahepatic portal twigs (Kaufmann). In many of the cases pus has been observed in the umbilical vein and the intrahepatic branches of the portal vein (Porak and Durante,[15] and Kaufmann [16] and in one of my cases). It is therefore not difficult to understand how the infection travels upward through the umbilical vein into the portal vein. From the portal vein the organisms are distributed throughout the liver. However, in other cases the umbilicus itself is unchanged, but on careful examination phlebitis is found farther along the veins (Porak and Durante [15]). In many cases the umbilicus and umbilical vein are grossly unchanged, but the vein may act as the portal of entry, even when the trunk is not involved (Porak and Durante [15] and Friedlander [17]). Porak and Durante emphasized the umbilical vein as the cause of umbilical sepsis. Runge,[18] on the other hand, pointed out the importance of the umbilical arteritis in umbilical sepsis. Among 55 infants seen in the obstetric clinic of the Charity Hospital who died of umbilical infection, he found arteritis in 54. Theoretically, it is possible to have abscess of the liver resulting from arteritis, the infected material being discharged into the hypogastric arteries and distributed throughout the body by the systemic circulation. In practically all cases, however, the infant dies from other septic complications, such as pneumonia, before a definite abscess of the liver develops.

How is phlebitis or arteritis brought about in these cases? The simplest explanation is that the infection is directly implanted into the umbilical vein or artery at the end of the ligated stump, the vein often being open, as has already been explained. Another explanation is as follows: The organism may be implanted in the Wharton jelly of the umbilicus, which becomes infiltrated with serous fluid. The process often extends into the perivascular tissue, and the vessel itself is involved. The inflammatory infiltration of the wall of the vessel causes paresis of the muscularis, which in turn causes dilatation of the vessel and thus may give rise to a thrombus, which may subsequently break down (Runge [18]). The infected thrombus then leads to arteritis or phlebitis. Runge stated that the thrombus and its disintegration are a secondary manifestation.

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15. Porak and Durante: Arch. de méd. d. enf. **4**:321, 1901.

16. Kaufmann, E.: Lehrbuch der speziellen pathologischen Anatomie für Studierende und Aerzte, ed. 8, Berlin, G. Reimer, 1922, vol. 1, p. 720.

17. Friedlander, A.: Acta paediat. **6**:431, 1926.

18. Runge, H.: Die Krankheiten der ersten Lebenstage, Stuttgart, Ferdinand Enke, 1893, p. 56.

Other methods of spread are by direct extension through the structures about the umbilical vein and the falciform ligament of the liver, by the lymphatic plexus and by the fine anastomosing vessels of the thin falciform ligament. The falciform ligament, if large, covers the entire right lobe of the liver and is adherent to the posterior surface of the peritoneum of the anterior abdominal wall. A capillary space is thus formed between the ligament and the peritoneum and between the ligament and the liver. The small vessels shining through the peritoneum of the falciform ligament have direct communication with the posterior surface of the anterior abdominal wall and the capsule of the liver. The lymphatics of the falciform ligament also have direct communications with the retrosternal or anterior mediastinal and peribiliary lymph nodes. In infections of the umbilicus the peritoneum which is in contact with the skin may become easily involved, and localized peritonitis may readily occur. If the infective organism is of moderate virulence the peritoneum may become plastered to the capsule of the liver and to the thin triangular falciform ligament. The inflammatory process then extends by lymphatics or blood vessels into the parenchyma of the liver and forms an abscess of the liver, or it extends by capillary attraction over the posterior surface of the anterior abdominal wall and capsule of the liver and forms an extracapsular abscess. If the micro-organisms are very virulent the infection spreads so rapidly that the host is unable to offer enough resistance to localize it. In such cases peritonitis rapidly develops, and the infection also travels along the lymphatic drainage system to the abdominal wall, scrotum, urachus, umbilical arteries and vein and to the diaphragm and mediastinum. Death occurs in from one to three days from septicemia, as a rule.

It is peculiar to note that the majority of the abscesses of the liver from umbilical infection occur in the right lobe. The predilection of the right lobe to abscess can be explained by the following facts: First, the anatomic relation of the falciform ligament to the liver is a factor. Second, the infective organisms are more apt to be thrown into the superior mesenteric stream (which flows into the right lobe of the liver) because of the frequency with which the umbilical vein joins the right side of the portal vein. Third, the right lobe of the liver is many times larger than the left lobe; therefore, it receives a larger number of organisms than the left. Fourth, the right hepatic vein makes an obtuse angle with the portal vein, so that the direction is more in a straight line in comparison with the left hepatic vein, the portal vein and the umbilical vein. As a result the current of flow is more likely to go into the right lobe of the liver than into the left, in analogy to the lodging of a foreign body in the right bronchus.

It is interesting to note in conjunction with abscess of the liver in new-born infants that many cases of necrosis of the liver were reported

in which the bacteriologic findings were different from those in cases of abscess of the liver. Amsler [19] described a case of miliary necrosis of the liver and adrenal glands but was unable to find the organism in the necrotic tissue. He expressed the opinion that he was dealing with the effects of a toxin. Bender,[20] Oppenheim [21] and Lonicier [22] have described similar lesions in reports of cases of congenital syphilis. Aschoff [23] and Henle [24] observed gram-positive, argentophilic, rodlike organisms. With Levaditi's stain Schneider [25] observed argentophilic, rod-shaped bacilli. Schwarz [26] saw similar argentophilic rods in material from one of his patients with active omphalitis. Kantschewa,[27] on the other hand, detected argentophilic spirochete-like organisms, but not those of syphilis. In his other case Schwarz [26] noted gram-positive diphtheroid organisms. MacMahon and Mallory [28] observed hemolytic streptococci in one of the infants with active omphalitis.

COMMENT

As far as I am aware, a solitary leaflike abscess like that observed in my first case has never been described before as occurring in an infant. Beaver,[29] in 1931, described several cases of multilocular leaf-shaped abscess of the liver in adults secondary to abscess in the head of the pancreas and perforated duodenal ulcers. The organisms observed in these cases were predominantly of the Staphylococcus pyogenes group, associated with other organisms. In my case Staphylococcus pyogenes-citreus was found in association with B. coli-communis. In Beaver's cases the localization of the right lobe can be easily explained by the present conception of the bilaterality of the liver (Serge [30] and McIndoe and Counseller [31]), the right and left lobes of the liver having definite areas of drainage from the mesenteric veins.

Concerning my first case, I believe that the infection became localized in the right lobe of the liver through the lymphatics about the

19. Amsler, C.: Centralbl. f. allg. Path. u. Anat. **23**:817, 1912.
20. Bender, H.: Virchows Arch. f. path. Anat. **177**:44, 1912.
21. Oppenheim, R.: Virchows Arch. f. path. Anat. **182**:237, 1905.
22. Lonicier, M.: Beitr. z. path. Anat. u. z. allg. Path. **39**:539, 1906.
23. Aschoff, L.: Verhandl. d. deutsch. path. Gesellsch. **4**:178, 1901.
24. Henle, quoted by Schwarz, L.: Virchows Arch. f. path. Anat. **254**:203, 1925.
25. Schneider: Virchows Arch. f. path. Anat. **219**:74, 1915.
26. Schwarz, L.: Virchows Arch. f. path. Anat. **254**:203, 1925.
27. Kantschewa, M.: Ztschr. f. Kinderh. **34**:169, 1925.
28. MacMahon, H. E., and Mallory, F. B.: Am. J. Path. **7**:299, 1932.
29. Beaver, D. C.: Am. J. Path. **7**:259, 1931.
30. Serge: J. de méd. de Bordeaux **22**:327, 1902.
31. McIndoe, A. H., and Counseller, V. S.: The Bilaterality of the Liver, Arch. Surg. **15**:589 (Oct.) 1927.

umbilical vein and along the falciform ligament, first, because the lumen of the umbilical vein, although narrowed, did not show evidences of phlebitis and, second, because the round ligament was the center of the intra-abdominal abscess. The suppurative process had extended into the thin falciform ligament, which covered the right lobe of the liver and had traveled along the mesenteric attachment of the umbilical vein and the falciform ligament to the posterior surface of the anterior abdominal wall. The thin, broad falciform ligament was shaggy and moth-eaten by the suppurative process. Externally, the umbilical cicatrix appeared normal, but the peritoneum on the abdominal wall was injected.

Another possibility in this case, suggested because of the subacute nature of the abscess, is that the infective organism may have been discharged into the portal vein through the umbilical vein probably early in life, a few days after birth, and may have left no trace of inflammation in the umbilical vein. Such a condition has been reported by Porak and Durante.[15] Since Staphylococcus pyogenes, both hemolytic and nonhemolytic, is present only in the first seven to eight days of life in about 20 per cent of the cases (Küstner [11]), early infection is plausible.

In the second case it is interesting to note that the abscess was located on the surface of the right lobe of the liver and that another abscess was present in the intra-abdominal region, with the round ligament as its center. Microscopic sections of the umbilical vein showed that the lumen contained a moderate amount of vascular connective tissue, but outside of the wall there were a few groups of pus cells. As the wall of the vein approached the abscess it became densely infiltrated with leukocytes, which suggests to me the possibility that the endophlebitis seen grossly may have been not primary but secondary to the periphlebitis. In this case the infection may have traveled about the structures of the umbilical cord and vein by the lymphatics of the falciform ligament and the periumbilical lymphatics or by direct continuity. The presence of an extracapsular abscess of the liver is in favor of this contention.

Another fact that struck me was the presence of pyogenic staphylococci in the abscess in each case. In these instances, because of a lessened virulence of the organisms the infant did not succumb early, and sufficient time, therefore, elapsed for the development of the abscess. The great rarity of this type of infection in infants can be seen by the fact that only 7 similar cases have been previously reported.

The reports of these 7 cases of abscess of the liver of umbilical origin were found in a complete review of the literature in which 145 cases of abscess of the liver in children were discovered. This series included (in addition to the 7 cases in which the abscess was of umbilical

origin) 17 cases in which it was of metastatic origin, 43 in which it was of dysenteric origin, 6 in which it was due to typhoid, 16 in which it followed appendicitis, 17 in which it was due to intestinal worms (especially Ascaris and Echinococcus), 11 in which it was of tuberculous origin and 2 in which it followed influenza. Among the remaining cases, trauma was listed as the causative factor in 19 instances, and 7 cases could not be classified.[32]

Of the cases listed, 24 were those of infants under 2 years of age. Besides the 7 cases just mentioned, in which the abscess was of umbilical origin, there were 8 cases in which it was of dysenteric origin, 4 in which it was of metastatic origin, 2 in which it was due to trauma and 3 in which it was due to each of the following, respectively: tuberculosis, influenza and infection caused by Ascaris lumbricoides. Incidentally, it may be mentioned that despite recent work [33] showing the great frequency of infection due to Ascaris in some countries, abscess of the liver from this cause is rare. It is also interesting to note that all the cases in which the abscess was of dysenteric origin occurred in warm countries—India, Egypt and South America—while those in which it was of umbilical origin occurred in countries with a temperate climate —Germany, England, Norway and France.

Analyzing over 4,000 postmortem examinations performed in a four year period at the Cook County Hospital, I found reports of only 4 other cases of abscess of the liver in children. These occurred in children aged 2½, 5, 14, 15 years, respectively, and the abscess was not of umbilical origin. In the same series of autopsies abscess of the liver was observed in 22 adults. Generally speaking, the common causes of this condition in adults (amebic dysentery, colitis, suppurative appendicitis and cholecystitis) are rare in children.

Abscess of the liver of umbilical origin can be divided into three types on the basis of the symptomatology and morbid anatomy. These are the subacute type, with a localized abscess; the acute type, with diffusely scattered miliary abscesses, and the fulminating type, with multiple necrotic processes or abscesses of the liver as a part of septicemia. The first two types are generally due to a staphylococcus, while the causative organism in the third is generally a streptococcus. The 2 cases described here belong in the first group. The symptoms and post-

32. Because of lack of space it has been found impossible to include the entire list of 145 cases. Any one interested can obtain the list of references from me. Most of the older cases in this review were reported by Musser (Cyclopedia of the Diseases of Children, edited by John M. Keating, Philadelphia, J. B. Lippincott Company, 1890, vol. 3, pp. 466-487) and LeGrand (Arch. de méd. d. enf. **9**:129, 1906).

33. Horilleno, F. S., and Haughwout, F. G.: Philippine J. Sc. **16**:1, 1920. Hoffmann, W.: Monatschr. f. Kinderh. **16**:82, 1919.

mortem observations can be taken as typical of this group of cases. Clinically, the condition must be differentiated from melena neonatorum, catarrhal gastro-enteritis and intestinal obstruction. The prognosis in these cases of abscess of the liver secondary to omphalitis is poor, the only hope for recovery being in surgical intervention. For this a two stage operation is generally recommended.

Thus, it must be borne in mind that umbilical infection of low virulence may cause serious damage, and every effort should be made at the time of birth to prevent the occurrence of these apparently harmless and often symptomless infections.

SUMMARY

Two cases of abscess of the liver in 6 week old infants, secondary to umbilical infection, are described, with the postmortem observations. In both cases the infection entered shortly after birth and gave no local signs at or about the umbilicus.

As far as could be determined, a solitary leaflike abscess of the liver, like that in case 1, has never before been reported in a young infant.

The embryology and histology of the umbilicus are discussed briefly as an aid to understanding the pathogenesis of abscess of the liver following umbilical infection of low virulence.

Reports of only 7 similar cases could be found in a review of the literature.

INTRAPERICARDIAL TERATOMA

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AND
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The occurrence of mediastinal teratoma in children is unusual. Hedblom [1] in a recent review of the literature found reports of 191 cases of intrathoracic dermoid cyst and teratoma. Of these only 21, or 11 per cent, occurred in children under 12 years of age; 6 occurred in children less than 1 year of age.

The following report concerns an infant with a teratoma completely enclosed within the pericardial sac. Since only 2 similar cases are on record, it was thought worth while to report this one.

REPORT OF CASE

E. C., a white girl 3 weeks old, was admitted on Sept. 13, 1933, to the Strong Memorial Hospital, where she died on September 22.

Present Illness.—Cough and dyspnea had been present since birth. The cough was intermittent and at times spasmodic. She took breast feedings well and thrived the first week of life. When 6 days old, she had three convulsions each of which persisted for approximately two minutes. Because of these she was taken to a hospital. During the second week of life she began to have attacks of cyanosis. She also had a temperature of 102 F. and diarrhea for a few days. She received two transfusions of blood, the first on the tenth and the second on the fourteenth day of life. On several occasions she nearly expired during attacks of cyanosis and dyspnea.

Past and Family History.—The pregnancy was normal. The child was born at full term following a normal delivery. She breathed spontaneously, was not a "blue baby" and weighed approximately 10 pounds (4,530 Gm.). Neither cyanosis nor convulsions occurred immediately after birth. The infant was fed at the breast for one week; after this she received a diet of evaporated milk. She received no cod liver oil or orange juice. Except for a slight loss immediately after birth, she gained weight steadily.

The mother was 23 and the father 26 years of age. Both were in good health. They had a daughter 3 years and a son 1 year old who had never been ill. The patient's grandmother had had "slight heart trouble."

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1. Hedblom, C. A.: Intrathoracic Dermoid Cysts and Teratomata, J. Thoracic Surg. **3**:22, 1933.

Physical Examination.—On September 13 the temperature was 38.8 C. (101.8 F.), the pulse rate 150 and the respiratory rate 30. The infant was extremely dyspneic and coughed incessantly. She was fairly well developed and nourished. The skin was warm. The face and mucous membranes were somewhat blue. The lymph nodes were not enlarged. The ears were normal. The pharynx was moderately injected, but no exudate was present. The sternum was prominent, especially the upper third. The respiratory expansion of the chest was normal. On percussion at the right of the sternum there was dulness which extended over the apex of the right lung. The cardiac dulness on the left was within normal limits. Breath sounds over the area of dulness were distant. A few transient râles were heard. The heart sounds were rapid, regular and indistinct. No murmurs were heard. The pulmonic second sound was accentuated. The blood pressure could not be obtained in either the arms or the legs. The abdomen was distended and soft. The border of the liver extended 2 fingerbreadths below the right costal margin and below the xyphoid process. The spleen was not palpable. No masses were present. The genitals were normal. The extremities showed no clubbing or edema. The results of neurologic examination were negative.

Laboratory Findings.—Examination of the blood showed: hemoglobin, 110 per cent (Sahli); red blood cells, 4,220,000; white blood cells, 15,400; polymorphonuclears, 78 per cent; polymorphonuclear eosinophils, 1 per cent; lymphocytes, 19 per cent; monocytes, 2 per cent; smear, normal. The urine was normal. The tuberculin test (human and bovine 1:1,000), the Schick and Dick tests and the cultures of the nose and throat for Klebs-Löffler bacilli were negative. The blood calcium was 10.4 and the phosphorus 5.6 mg. per hundred cubic centimeters. A roentgenogram of the chest showed great enlargement of all the chambers of the heart; that of the left ventricle and right auricle was most striking. The electrocardiogram (after administration of digitalis) showed right ventricular preponderance, nodal rhythm and sinus arrhythmia.

Course.—The patient's condition on admission was extremely critical. She had two severe attacks of cyanosis and dyspnea. Oxygen with 5 per cent carbon dioxide was administered by means of a mask. Injections of caffeine and epinephrine were given, and artificial respiration was carried out. During her stay in the hospital, the attacks of cyanosis occurred several times both during and between feedings; each time the patient nearly died.

A presumptive diagnosis of idiopathic hypertrophy of the heart was made. The administration of digitalis was started on the night of admission, a total of 1.75 cc. of the tincture being given. The following morning the pulse rate was 76, and there was evidence of a partial intermittent heart block. On September 19 the patient's general condition became worse. She also presented marked stridor, which had not been observed before. A needle was inserted in the second intercostal space about 3 cm. lateral to the right border of the sternum and directly posteriorly. The needle apparently passed through solid tissue which did not feel like lung. A small amount of very dark blood was aspirated. It was concluded that a solid tumor was present. The child was given 1 dose of high voltage roentgen therapy, ⅓ erythema dose (180 roentgens). On September 22 the fluoroscope clearly demonstrated a dense shadow superimposed on the heart and practically covering it. The shadow did not pulsate, although the margin of the heart could be seen pulsating. The child became progressively worse and died on September 22. The diagnosis was mediastinal tumor.

Pathologic Examination.—External examination of the baby revealed nothing significant except a marked convexity of the sternum. When the chest plate

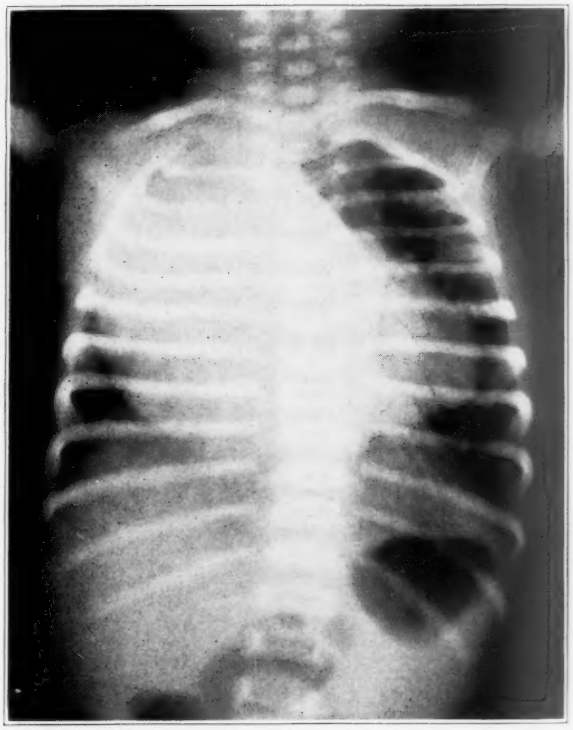

Fig. 1.—An anteroposterior view of the chest which shows the increased cardiac shadow.

Fig. 2.—Gross appearance of the tumor which is situated adjacent to the right border of the heart.

was removed, the anterior mediastinum was seen to contain a mass, which proved to be within the pericardial sac. The pericardium, especially over the mass, was gray and somewhat thickened. When the pericardium was opened, a tumor measuring 5 cm. in diameter was exposed. It was firmly adherent to the parietal pericardium on the one hand and on the other to the wall of the right ventricle, to the right auricle at the base of the great vessels and to the transverse aorta. It overlay the bifurcation of the trachea, but there was apparently no distortion of the lumen. The pericardial sac contained about 20 cc. of clear yellowish fluid.

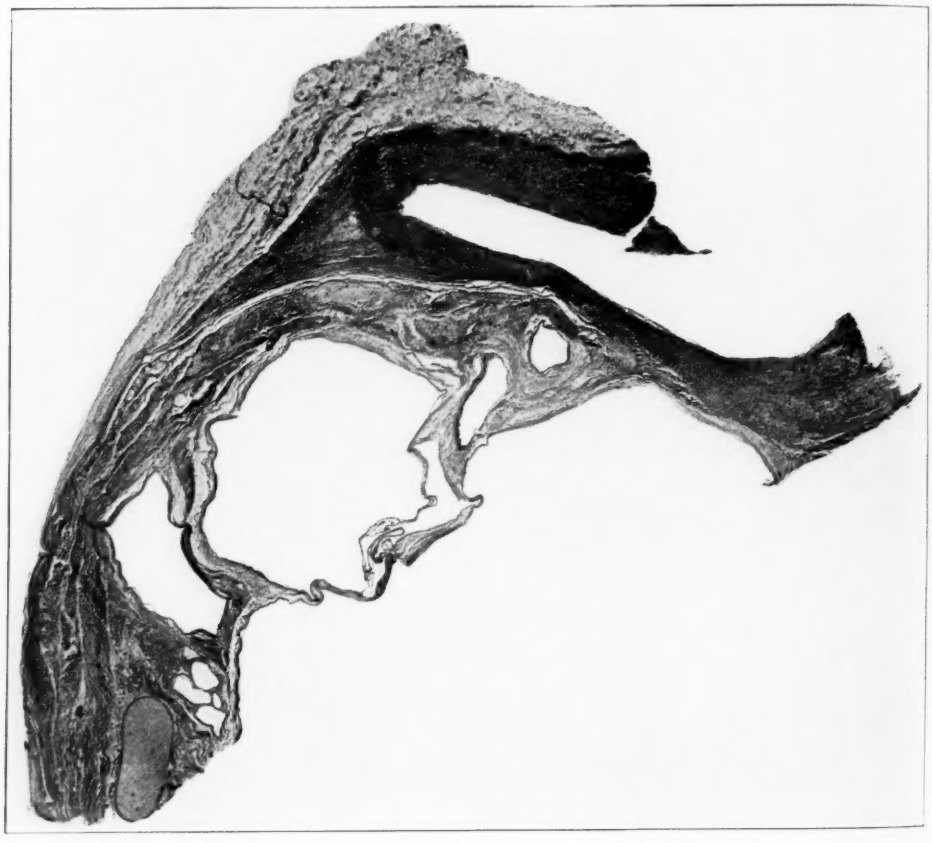

Fig. 3.—Photomicrograph showing the relation of the tumor to the adventitia of the aorta.

The heart was not enlarged and was hanging from the mass as a small appendage. There was a small area of epicardium at the apex of the left ventricle which had a roughened granular appearance, and there were a few fine fibrinous adhesions binding this area to the pericardium. The mass was firm in consistency, but transillumination was not successful; on gross section it presented a multilocular cystic appearance similar to that of a multilocular cystic ovary. The cysts varied in diameter from several millimeters to 2 cm. The capsule and walls of the cyst were composed of what appeared to be gray fibrous tissue, and there was very little stroma between the cysts. The cavities contained a thick, more or less clear.

glairy mucoid material, and the stroma appeared to be avascular. On section, the heart showed nothing significant except a functionally patent foramen ovale and ductus arteriosus. The mass did not infiltrate the wall of the heart or communicate with any of the chambers or vessels and did not alter the normal conformation of the heart. The thymus was very small. The rest of the organs were normal.

Microscopic Examination of Tumor.—The capsule was thin and composed of fibrous tissue which was continuous with the adventitia of the aorta. The sections showed numerous cysts, some of which were lined with simple columnar, stratified columnar or ciliated columnar epithelium. Others were lined with a single layer of cuboidal epithelium and a few with transitional epithelium. One cyst contained a papillomatous structure covered with epithelium similar to that enveloping the choroid plexus. Between these cysts there were smooth muscles, cartilage and glandular structures, many of which resembled pancreas. No islets of Langerhans were seen. Much of the columnar epithelium contained a pink-staining material. Microscopic examination of the other organs revealed nothing remarkable.

Anatomic Diagnosis.—The diagnosis was intrapericardial cystic teratoma, functionally patent foramen ovale and ductus arteriosus.

COMMENT

The signs and symptoms of mediastinal tumor vary a great deal depending on the size and location of the tumor and the amount of pressure on adjacent organs. In this case cough, dyspnea and cyanosis were the principal symptoms. On physical examination, the facial edema, protrusion of the wall of the chest and dulness on percussion over the area of the tumor associated with distant breath sounds were suggestive of mediastinal tumor. The possibility of mediastinal tumor became more evident when a solid mass was encountered by the needle on attempted aspiration. When fluoroscopic examination disclosed a dense shadow which did not pulsate superimposed on the heart, this opinion was confirmed.

The unusual location of the tumor is of great importance from both the diagnostic and the pathologic point of view. It is of interest to note that the provisional diagnosis of idiopathic cardiac hypertrophy was also made in the case previously reported by Grimm.

In the literature on the subject of mediastinal tumors, the dermoid cyst and the teratoma are considered together. Those tumors that contain derivatives of ectoderm and mesoderm are described as dermoid cyst, while those containing derivatives of all three germ layers, ectoderm, endoderm and mesoderm, are designated as teratoma.

The exact genesis of the teratoma is obscure. Ewing [2] attributed the origin to aberrant sexual cells and stated that this may account for the predilection of teratoma for the sexual glands. Extragenital tera-

2. Ewing, J.: Neoplastic Diseases, ed. 4, Philadelphia, W. B. Saunders Company, 1934.

toma has been ascribed to the development of isolated blastomeres which undergo varying degrees of development (Marchand).

The two other recorded cases of a teratoma within the pericardial sac are of interest. The first was reported by Joel [3] in 1890 and occurred in a 14 year old boy. The tumor, which was cystic, was the size of a hen's egg and was attached to the adventitia of the pulmonary artery within the pericardium. The patient had bilateral cryptorchidism. The other case was that of a 3 month old infant reported by Grimm [4] in 1927. In this instance the tumor was also cystic and in intimate association with the adventitia of the great vessels. The provisional diagnosis in this case was idiopathic cardiac hypertrophy.

SUMMARY

A report of a case of mediastinal tumor in an infant is presented. The tumor was a cystic teratoma which apparently arose at the base of the great vessels and grew completely enclosed within the pericardium. The associated observations were a patent foramen ovale and ductus arteriosus.

3. Joel, J.: Ein Teratom auf der Arteria pulmonalis innerhalb des Herzbeutels, Virchows Arch. f. path. Anat. **120**:381, 1890.

4. Grimm, H. W.: Mediastinal Teratoma (Case Report), Radiology **8**:438, 1927.

Progress in Pediatrics

DERMATORRHEXIS

WITH DERMATOCHALASIS AND ARTHROCHALASIS (THE SO-CALLED EHLERS-DANLOS SYNDROME)

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The so-called Danlos or Ehlers-Danlos syndrome consists of three symptoms: (1) pronounced fragility of the skin and its blood vessels, with breaking, splitting and the formation of hematomas and pseudotumors subsequent to the slightest trauma (dermatorrhexis-fragilitas cutis), (2) a more or less pronounced hyperlaxity and hyperelasticity of the skin (dermatochalasis-laxitas cutis) and (3) more or less pronounced hyperlaxity or hyperflexibility of the joints (arthrochalasis-laxitas articularis).

I have had the opportunity of observing three patients with this syndrome, and I wish to report their cases, not to add much to the descriptions of this subject which have appeared in the European literature and in the paper by Tobias [1] in this country but because it seems to me that little attention has been paid here to this syndrome by dermatologists, pediatricians and orthopedic surgeons. Furthermore, I think that the terminology needs revision, so that the importance of one of the symptoms (the fragility of the skin) over the others will be stressed.

After one has observed a single case of dermatorrhexis, the diagnosis can be made almost at first sight. When the patient, most often a child, uncovers the knees and shins, a characteristic picture is presented to the eye of the examiner, a picture of wrinkles, hyperpigmentations, depigmentations, shiny lines and flabby, soft, raisin-like growths among islands of normal skin. A similar appearance to a lesser degree is present on the elbows and even on the head. Where bones are but little protected by subcutaneous tissue (elbow, knee, shin, ankle, forehead, zygoma, chin), regions commonly hit against hard objects, there are disfiguring scars (fig. 1).

From the Department of Dermatology, the Rhode Island Hospital.

1. Tobias, N.: Danlos Syndrome Associated with Congenital Lipomatosis, Arch. Dermat. & Syph. **30**:540 (Oct.) 1934.

Palpation of the skin reveals a peculiar velvety texture. The skin glides easily over the bones and muscles. The condition may be described as the opposite of scleroderma. The subcutaneous fat seems to have disappeared so that the bony prominences are readily felt. If

Fig. 1 (cases 1 and 2).—The scars and molluscoid raisin-like tumors on the elbow, knees and shins (dermatorrhexis).

one has the opportunity of seeing a patient soon after he has bumped his leg or head against a hard object, a wound will be seen, as if made by a cutting instrument, and the parents will tell the examiner that it is of no avail to put in stitches, since they will not hold. Adhesive tape must be used to hold the edges of the wound together (fig. 2).

If the patient's skin is picked up between the index finger and the thumb, it will be found to stretch easily three or four times more than it will in a normal person (fig. 3). Examination of the joints reveals

Fig. 2 (case 1).—Splitting of the skin as it appeared one week after the part had been bumped against a hard object (dermatorrhexis).

hyperflexibility, especially marked in the fingers and toes, which frequently can be bent backward so as almost to touch the dorsum of the hand or foot (fig. 4).

In the cases described by Margarot and his associates [2] the extension of the knees gave the impression of a tabetic posture. Ehlers [3] described a case in which the skin was similar to the peeling of a baked apple and loose enough to be wrapped around a finger one and one-half times. This patient complained of spontaneous dislocation of the patella, which would force him to stop while walking and replace the bone. These signs are sufficiently definite to make the syndrome a clinical entity.

REPORT OF CASES

CASE 1.—R. K., a 10 year old white boy, was first seen at the dermatologic clinic of the outpatient department of the Rhode Island Hospital in October 1932. He weighed 42 pounds (19 Kg.) and was 43½ inches (110 cm.) tall.

The family history was not important. The boy was a premature (8 months) infant, weighing 4 pounds (1,815 Gm.) at birth. Delivery was instrumental. The

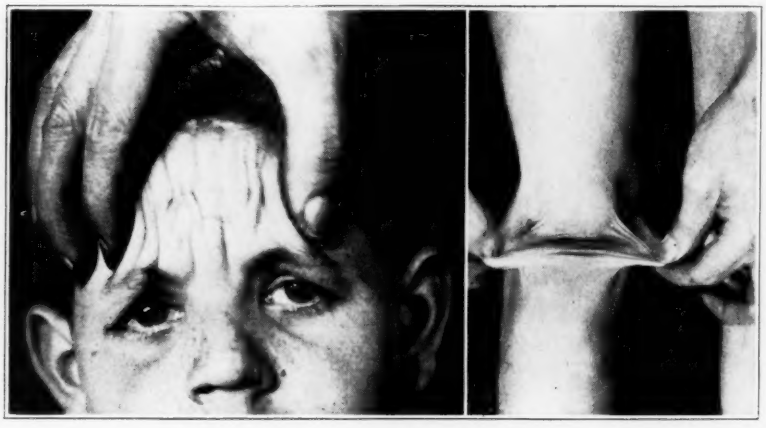

Fig. 3.—The scars on the forehead in case 1 and the dermatochalasis in cases 1 and 3.

infant was breast fed for six months. Dentition began at 6 months, walking at 1 year and talking at 18 months.

He had had measles, whooping cough and chickenpox. No abnormalities were noticed until the child was 3 years old. Then it was observed that whenever he fell or hit any part of the body against a hard object the skin over the injured area split wide open without bruising. The bleeding was moderate. Attempts at suturing failed because the stitches would not hold. The only successful method was the use of adhesive tape.

The skin gave a peculiar velvety feeling to the touch. The knees and shins were covered with scars one on top of the other (fig. 2), and to a lesser degree

2. Margarot, J.; Deveze, P., and Coll de Carrera: Hyperlaxité cutanée et articulaire (syndrome de Danlos) existant chez trois membres d'une même famille, Bull. Soc. franç. de dermat. et syph. **40**:277, 1933; L'hyperlaxité familiale de la peau et des articulations (syndrome de Danlos), Montpellier méd. **3**:133, 1933.

3. Ehlers, E.: Cutis laxa, Neigung zu Haemorrhagien in der Haut, Lockerung mehrerer Artikulationen, Dermat. Ztschr. **8**:173, 1901.

the forehead and chin also were scarred. The laxity of the skin could be seen on the forehead, where it gave a picture similar to that of cutis frontis gyrata (fig. 3). A split on one leg had been the result of a bump on the edge of a staircase. A split over the right zygomatic bone was due to bumping against a door (fig. 2).

The patient was seen again in April 1935 and in December was admitted to the hospital for further study.

The Wassermann reaction of the blood was negative. The urine was normal. The hemoglobin content of the blood was between 80 and 88 per cent. A cell count showed 5,100,000 erythrocytes, 9,000 leukocytes, 69 per cent polymorphonuclears, 30 per cent lymphocytes and 1 per cent eosinophils. The number of platelets was diminished. The urea, sugar, cholesterol, chloride and phosphorus values were found to be normal on several occasions. The calcium content was 6.9 mg. per hundred cubic centimeters at the patient's first admission to the hospital. At

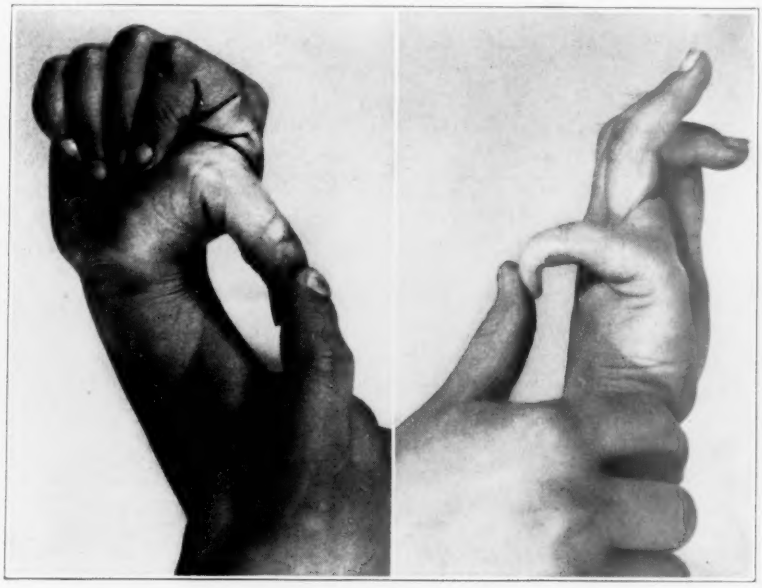

Fig. 4 (cases 1 and 2).—Arthrochalasis.

successive admissions it was 10 and 11.5 mg., respectively. The coagulation time was eight minutes, and the bleeding time was five and one-half minutes. Hemolysis began with a 0.3 per cent and was complete with a 0.45 per cent solution of sodium chloride. The van den Bergh reaction was negative. The icterus index was 6.

Injections of pilocarpine hydrochloride, solution of posterior pituitary, thyroid and parathyroid extract failed to show any abnormal response of the vegetative nervous system.

The basal metabolic rate averaged minus 6. The reaction to tuberculin was positive.

Roentgenograms of the entire body revealed no abnormality. A psychometric examination revealed an average intelligence. The patient showed a slight speech defect.

In April 1935 the weight was 51 pounds (23 Kg.), and the height was 4 feet and ¼ inch (123 cm.).

The therapy consisted of calcium, parathyroid extract, cod liver oil, viosterol and brewer's yeast.

Biopsy specimens were taken from various old and recent scars and from raisin-like molluscoid tumors. The pathologic report (Dr. B. E. Clarke) read as follows:

"The epidermis appears to be cornified, markedly waved and ridged. There are brown granules in the cytoplasm of the basal cells of the epidermis. The dermis consists of coarse and fine bundles of acidophilic fibrils which tend to follow the contour of the epidermis. The tissue is relatively loose, and some bundles of fibrils are wavy. The cellularity varies considerably, and there are scattered single and clumped mononuclear cells, as well as numerous coil glands, occasional hair follicles and some capillaries. The cytoplasm of these mononuclear cells is not vacuolated. In one section there are nodules composed of large fusiform cells, polyhedral mononuclear cells and large multinucleated giant cells with an abundance of acidophilic cytoplasm. There are extravascular erythrocytes in the dermis. The subdermis consists of rather vascular loose connective tissue and fat (fig. 5). Staining for elastic tissue shows in places in the dermis, but more especially in the subdermis, a dense reticulum of coarse and fine wavy elastic fibrils."

CASE 2.—V. C., a 9 year old American girl of Portuguese extraction, was seen at the dermatologic clinic of the outpatient department of the Rhode Island Hospital in March 1935 and subsequently was admitted to the hospital for study. She weighed 63 pounds (28.6 Kg.) and was 43 inches (109 cm.) tall.

The parents were first cousins. The mother was 43 years old and the father 45. The mother had had one miscarriage. She had had seventeen children, ten of whom were living. The patient was the thirteenth child. The whole family was suffering from malnutrition. Apparently the children were not given any fruit or green vegetables in their diet, and the milk they drank was from a cow fed only on hay.

The parents stated that for the past six years the patient bruised easily and that large blue-black swellings developed. When she was cut or bruised, bleeding lasted for a long time. The "cysts" due to bruises occasionally broke down, discharging a dark red jelly-like clot and fluid blood.

The general physical examination and laboratory tests revealed no abnormalities. A tuberculin test was not made. A psychometric test was not made, but the girl's intelligence seemed to be average.

A biopsy specimen from one of the molluscoid empty raisin-like tumors on the elbow (fig. 1) yielded the same result as in case 1, with a similar grouping of foreign body giant cells.

CASE 3.—W. J., a 10 year old American boy, was admitted to the fracture service of the Rhode Island Hospital for a fractured right forearm.

I was asked to see him because of the condition of the skin. With the experience gained from previous cases the house officers had already made the correct diagnosis.

The tuberculin test showed positive results. The basal metabolic rate was minus 22. The rest of the laboratory tests and examinations revealed no abnormalities. A psychometric test was not performed, but the intelligence seemed to be average. A biopsy was not made.

Since the publications of Ehlers [3] in 1901, Cohn [4] in 1907 and Danlos [5] in 1908, not much has been written on the subject of dermatorrhexis. Only one paper has appeared in the American literature.[1]

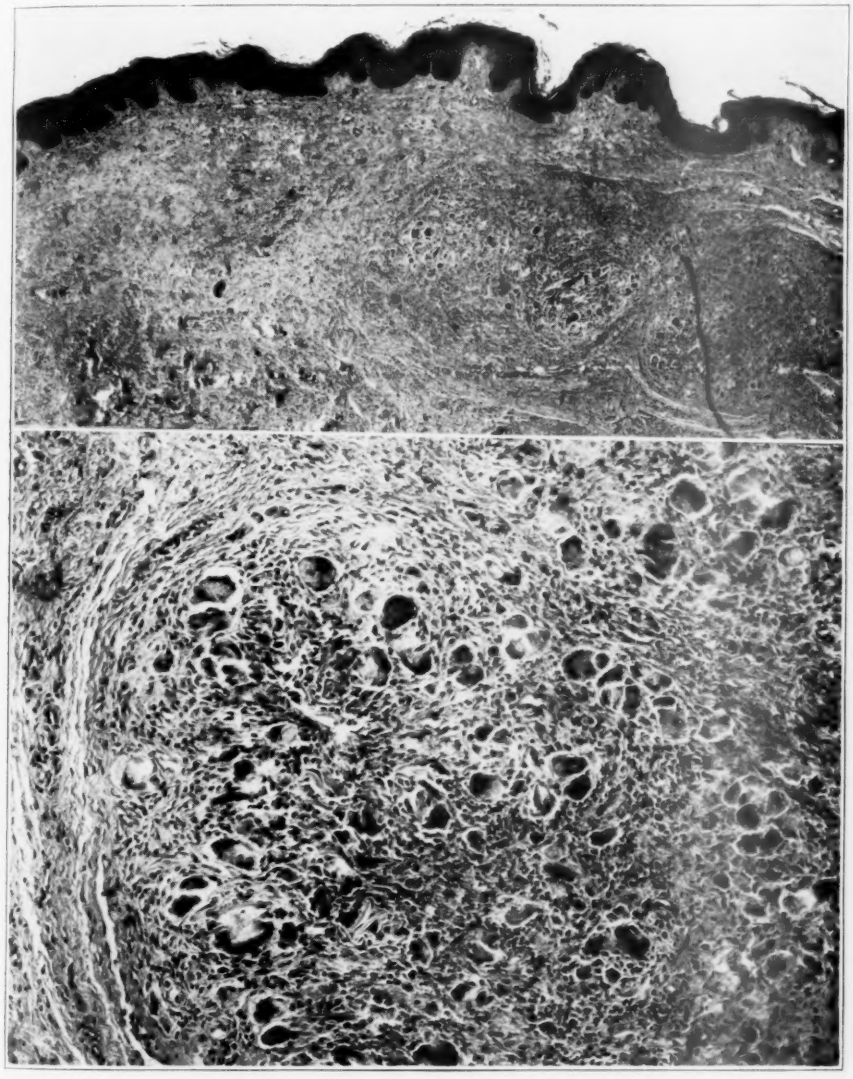

Fig. 5.—Photomicrographs of sections through a molluscoid raisin-like tumor showing the thinned and wavy epidermis and the group of foreign body giant cells.

4. Cohn, P.: Demonstration eines Patienten mit Gummihaut (cutis laxa) und eigentümlichen zirkumskripten Hautveränderungen, braunroten, eindrückbaren Erhebungen, Verhandl. d. deutsch. dermat. Gesellsch. **9**:415, 1907.

(Footnote continued on next page)

Part of the syndrome (the hyperelasticity and hyperlaxity) has been described in the past, but under various denominations, viz., dermatolysis, cutis laxa, cutis hyperelastica, loose skin, chalazoderma or chalasoderma, cutis pendula, elastic skin, india rubber skin, gummihaut. In the "Standard Classified Nomenclature of Disease" [6] the condition is listed under dermatolysis, cutis laxa.

Cases of circus "rubber men" have been reported, like that of the Spaniard who was able to draw the skin of the right pectoral region to the left ear, the skin of the chin onto the chest like a long beard and the skin of the right knee up and down for half a yard (Turner,[7] Burrows [8]).

Apparently no one before Ehlers [3] and later Cohn [4] and Danlos [5] considered, in addition to the hyperlaxity and hyperelasticity of the skin, the fragility of the skin, which is the cause of the splitting, the intracutaneous hemorrhages and the disfiguring scars and pseudotumors (dermatorrhexis).

Dermatorrhexis, as well as the entire syndrome, is not mentioned in American textbooks of dermatology, although dermatolysis, cutis laxa and cutis hyperelastica are described. Only the most recent European textbooks discuss the subject. Steiner,[9] describing congenital abnormalities of the skin in the chapter headed "Cutis Laxa," showed a good illustration of a patient with elastic skin and spoke of several cases of this syndrome (Cohn, Dobroworskaja, Shubert, Hirszfeldwa, von Wiener, Capurro, Mierowsky). Then he described, "in addition," the frequency of the hematomas, tumors and scar formations and said that the term cutis laxa does not correspond to the facts, because attention is not directed to the fragility of the skin and the subsequent formation of hematomas, tumors and scars. Gaté,[10] under the heading "Hyperelasticité de la peau," although not mentioning the splitting of the skin, briefly said that in exceptional cases the laxity of the skin is

5. Danlos, H.: Un cas de cutis laxa avec tumeurs par contusion chronique des coudes et des genoux (xanthome juvénile pseudodiabétique de MM. Hallopeau et Macé de Lépinay), Bull. Soc. franç. de dermat. et syph. **19**:70, 1908.

6. The Standard Classified Nomenclature of Disease, New York, The Commonwealth Fund, 1935, p. 126.

7. Turner, cited by Sée, M., in Besnier, E.; Brocq, L., and Jacquet, L.: La pratique dermatologique, Paris, Masson & Cie, 1900, p. 698.

8. Burrows, A.: Epidermolysis Bullosa with Cutis Hyper-Elastica, Proc. Roy. Soc. Med. **25**:1319, 1932. (This article includes a reproduction of a picture of the Spaniard with "rubber skin.")

9. Steiner, K., in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1932, vol. 4, pt. 1, p. 78.

10. Gaté, J., in Belot, J., and others: Traité de dermatologie clinique et thérapeutique, Paris, Gaston Doin & Cie, 1933, vol. 1, p. 226; vol. 2, pt. 2.

complicated by abnormal fragility. He reproduced good illustrations from Schulmann and Lévy-Coblentz'[11] paper.

In addition to the already mentioned cases of Ehlers,[3] Cohn,[4] Danlos,[5] Tobias[1] and Margarot and his associates[2] (three cases) and those listed by Steiner[9] (six cases), cases of typical or complete dermatorrhexis with hyperlaxity of the skin and joints have been reported on by Weber[12] (brief description of four cases presented by English dermatologists before English dermatologic societies), Schulmann and Lévy-Coblentz,[11] Burrows,[8] Cockayne,[13] Sézary and his associates[14] (good illustrations of the molluscoid tumors of the knee, in this case, are published in the theses of Miget[15]), LeCoulant,[16] Rocher and his associates,[17] Keinig,[18] Baliña and Sicardi,[19] Kalz[20] and Sézary and Horowitz.[21]

The condition is undoubtedly uncommon, but I do not think it can be called exceptional, as some authors have said (Gaté,[10] Sézary and Horowitz[21]). Twenty-seven typical or complete cases are mentioned in the present paper, and I feel that more cases will be recognized when physicians are better acquainted with the syndrome.

I wish to call attention to the fact that while unusual elasticity of the skin and laxity of the joints may give the owner some advantages, such as the possibility of performing on the stage as an acrobat, con-

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11. Schulmann, E., and Lévy-Coblentz, G.: Hyperélasticité cutanée (cutis laxa) et laxité articulaire avec fragilité anormale de la peau et tumeurs molluscoïdes post-traumatiques (syndrome de Danlos), Bull. Soc. franç. de dermat. et syph. **39**:1252, 1932; Une forme rare de dystrophie cutanée: le syndrome d'Ehlers-Danlos, ibid. **40**:380, 1933.

12. Weber, F. P.: Chalasodermia or "Loose Skin," and Its Relationship to Subcutaneous Fibrous or Calcareous Nodules, Urol. & Cutan. Rev. **27**:407, 1927.

13. Cockayne, E. A.: Inherited Abnormalities of the Skin and Its Appendages, New York, Oxford University Press, 1933.

14. Sézary, A.; Azérad, E.; Miget, A., and Deparis: Syndrome d'Ehlers-Danlos, Bull. Soc. franç. de dermat. et syph. **40**:1287, 1933.

15. Miget, A.: Le syndrome d'Ehlers-Danlos, Thèse de Paris, Louis Arnette, 1933.

16. LeCoulant, P.: Hyperlaxité cutanée et articulaire avec fragilité anormale de la peau et tumeurs molluscoïdes chez un enfant de 13 ans (syndrome de Ehlers-Danlos), Gaz. hebd. d. sc. méd. de Bordeaux **55**:261, 1934.

17. Rocher, H. L.; Petges, G., and LeCoulant, P.: Hyperélasticité articulaire et hyperélasticité cutanée d'origine congénitale dystrophique (syndrome d'Ehlers-Danlos), Rev. d'orthop. **21**:673, 1934.

18. Keinig, cited by Rocher, Petges and Lecoulant.[17]

19. Baliña, P. L., and Sicardi, A.: Cutis hyperelastica-fragilidad cutanea y tumores, Rev. argent. de Dermat. **59**:144, 1935.

20. Kalz, F.: Cutis laxa als Symptom allgemeiner Stützgewebsschwäche, Arch. f. Dermat. u. Syph. **171**:155, 1935.

21. Sézary, A., and Horowitz, A.: Syndrome d'Ehlers-Danlos, Bull. Soc. franç. de dermat. et syph. **42**:1744, 1935.

tortionist or circus freak, the possessor of fragile skin has the disadvantage of constantly being obliged to be cautious and the burden of disfiguring scars and tumors, provoked by the slightest injury, which in a normal person would be the cause of nothing more than a quickly disappearing bruise.

Hence, the peculiar fragility of the skin, from a dermatologic and practical standpoint, is the main feature of the syndrome.

The early case reports of Alibert,[22] and those of Vaglio,[23] Raspi [24] (fig. 6), Rocher,[25] Navarro Martín and Aguilera Maruri [26] and others

Fig. 6.—A 14 month old girl with dermatochalasis without dermatorrhexis and arthrochalasis (from Raspi [24]).

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22. Alibert, cited in Besnier, E.; Brocq, L., and Jaquet, L.: La pratique dermatologique, Paris, Masson & Cie, 1900, p. 695, fig. 129.

23. Vaglio, R.: Un caso di cutis laxa, Pediatria **31**:321, 1923.

24. Raspi, M.: Di un caso di "cutis laxa," Riv. di clin. pediat. **25**:648, 1927.

25. Rocher, H. L.: Une nouvelle dysmorphose articulaire congénitale: laxités articulaires congénitales multiples, in Livre jubilaire du Professeur Henri Hartmann, Paris, Masson & Cie, 1932, p. 553.

26. Navarro Martín, A., and Aguilera Maruri, C.: Un caso de cutis laxa y lesiones atróficas de tipo eritromiélico, Actas dermo-sif. **25**:126, 1932.

mentioned hyperlaxity of the joints or hyperelasticity and hyperlaxity of the skin or both but not dermatorrhexis.

How could the case report of Rocher,[25] with its beautiful illustrations of hyperlaxity of the skin and joints, but no splitting, scars or tumors, be called "one of the best cases ever described of the Ehlers-Danlos syndrome," as it was in the latest comment which I happened to read on the subject (Petges [27])?

Figure 5 from Raspi [24] showing cutis laxa without dermatorrhexis is reproduced to demonstrate the confusion of labeling as cutis laxa laxity of the skin and joints either with or without dermatorrhexis. By using the heading cutis laxa [28] the most important sign of this syndrome, the dermatorrhexis, is completely overlooked.

In regard to the term dermatolysis,[6] as lysis means to dissolve, and it is used, for instance, in orthopedic surgery to indicate the operative dissolution of a joint (arthrolysis), how can the same suffix used in the word dermatolysis indicate not a dissolution but simply a loosening of the skin? For the sake of clarity and simplicity in nomenclature I think the heading dermatorrhexis with or without dermatochalasis and arthrochalasis answers the purpose better than a proper name or any other lengthy name.

Various conditions have been found concomitant with the syndrome, viz., malformations of the appendages of the skin, epidermolysis bullosa, nevi, generalized lipomatosis and poor mental development.

In two of my cases and in one of Capurro's [29] the reaction to tuberculin was positive. In one of my cases the patient had been delivered by instrument prematurely and was physically underdeveloped. In my second case the parents were consanguineous, and the mother was weak owing to numerous confinements and malnutrition. In none of my cases were other members of the family affected, while von Wiener reported the condition in three generations and Margarot and his associates [2] in three members of the same family.

From the findings of various authors the pathologic changes seem to take place essentially in the derma: thinning, rarefaction and torsion of the collagenous fibers, as well as thinning and flattening of the whole cutis. The foreign body giant cells observed in two of my cases did not seem to have any particular significance; this finding was reported before only in the case of Sézary and his associates (the patient having also tuberculosis of the lungs).

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27. Petges, G.: A propos de la communication de MM. A. Sézary et Horowitz sur un cas de syndrome d'Ehlers-Danlos, Bull. Soc. franç. de dermat. et syph. **43**: 66, 1936.

28. Footnotes 6, 9 and 10.

29. Capurro, R.: Cutis laxa, hematomas, atrofias cutáneas, hipotonía muscular, laxitud articular, Arch. latino-am. de pediat. **20**:713, 1926.

My conclusion in regard to the etiology and pathology of this condition is that little is known concerning them and that one may consider constitutional mesenchymatosis, endocrine disturbance, atavism or an abnormal form of Recklinghausen's disease, although dermatorrhexis has never been reported in a case of Recklinghausen's disease. It will be well to call the condition idiopathic dermatorrhexis with idiopathic dermatochalasis and arthrochalasis.

The scarring which follows splitting of the skin bears some resemblance to striae cutis distensae or atrophicae, and I wonder if a similar mesenchymatic fragility could be the background of the so-called idiopathic striae cutis distensae or atrophicae, which may be found also in adolescent boys and girls. It has been demonstrated that these striae follow the lines of cleavage of the skin, and they are supposed always to be due to trauma, although perhaps unnoticed. They are found usually in weakened persons, after long illnesses, in persons with a tendency to visceral ptosis and in those with congenital dystrophic conditions (Nardelli [30]).

SUMMARY

The so-called Danlos or Ehlers-Danlos syndrome is characterized by: (1) pronounced fragility of the skin and its blood vessels, with subsequent splitting and the formation of hematomas and pseudotumors at the slightest provocation (dermatorrhexis, fragilitas cutis), (2) a more or less pronounced hyperlaxity and hyperelasticity of the skin (dermatochalasis, laxitas cutis) and (3) a more or less pronounced hyperlaxity of the joints (arthrochalasis, laxitas articularis).

Twenty-seven cases have been reported in the literature.

Since dermatorrhexis is the most important part of the syndrome from a dermatologic and practical point of view, it is suggested that proper names or lengthy names be avoided and the term dermatorrhexis with or without dermatochalasis and arthrochalasis be used.

The syndrome is well defined and constitutes a clinical entity to be included in the group of congenital dystrophic anomalies.

The etiology and pathology are obscure.

122 Waterman Street.

30. Nardelli, L.: Le "striae cutis atrophicae," Gior. ital. di dermat. e sif. **76:** 607, 1935; discussion by Crosti.

News and Comment

GENERAL

American Board of Pediatrics.—The annual meeting of the American Board of Pediatrics was held in Kansas City, Mo., on May 9 and 10, 1936. All the directors and officers were reelected for the ensuing year. Dr. Franklin P. Gengenbach was reappointed as a member of the Board from the Section on Pediatrics of the American Medical Association.

The Board has found it necessary to increase the number of examiners and voted to add a group of examiners who shall act as members of the Board for purposes of examination. Dr. T. B. Cooley and Dr. Horton R. Casparis were appointed at this meeting.

A total of 224 certificates were granted by examination and on record during the past year, and a total of 563 certificates have been granted since the organization of this Board.

The fee for examination and certification after Sept. 30, 1936, has been placed at $30. Attention has previously been called to the fact that the fee of $20 now charged by the Board of Pediatrics is much lower than that of the boards for other special fields. Experience gained in the past two years has shown that an increase in the fee is necessary to meet the expenses of the board. The increased fee is still lower than that charged by other boards (e. g., Board of Otolaryngology $50; Board of Dermatology $35; Board of Ophthalmology $50).

Physicians Certified by American Board of Pediatrics.—The following physicians have been certified by the American Board of Pediatrics since the last report:

John William Amesse, Denver; Maurice Lamm Blatt, Chicago; Jules M. Brady, St. Louis; M. Bernard Brahdy, Mt. Vernon, N. Y.; Chester R. Brown, Arlington, N. J.; George Walter Caldwell, New York; D. M. Cowie, Ann Arbor, Mich.; George Martin Guest, Cincinnati; Lewis Webb Hill, Boston; William W. Howell, Boston; Archibald L. Hoyne, Chicago; Herbert F. Jackson, New York; Mildred Wicker Jackson, Oyster Bay, N. Y.; George Kahn, Boston; John S. McDavid, Oak Park, Ill.; Morley D. McNeal, Highland Park, Ill.; William Diuguid Mims, Memphis, Tenn.; Dorothea May Moore, Cambridge, Mass.; Karlton G. Percy, Boston; Bret Ratner, New York; Aldert Smedes Root, Raleigh, N. C.; James Earl Russell Jr., Denver; D. Lesesne Smith Sr., Spartanburg, S. C.; Chester A. Stewart, Minneapolis; Thomas Durland Van Orden, New York; Arthur E. Wade, Seattle; John Zahorsky, St. Louis.

None of these was examined.

SOCIETY NEWS

Fourth International Congress of Pediatrics.—The Fourth International Congress of Pediatrics, to be held in Rome, has been postponed to the first week of April 1937.

Abstracts from Current Literature

Pathology, Bacteriology and Biochemistry

THE POSTERIOR LOBE OF THE PITUITARY GLAND; ITS RELATIONSHIP TO THE STOMACH AND TO THE BLOOD PICTURE. E. C. DODDS, G. M. HILLS, R. L. NOBLE and P. C. WILLIAMS, Lancet **1**:1099 (May 11) 1935.

In a previous study it was shown that an extract of the posterior lobe of the pituitary gland when injected subcutaneously into animals produces an acute hemorrhagic lesion of the acid-bearing area of the stomach. It was further shown that if the contents of the stomach are rendered alkaline prior to the injection it is impossible to obtain the characteristic reaction. The most significant points concerning the gastric lesion are, first, its extremely accurate limitation to the area of the stomach in which oxyntic cells are found and, second, the complete absence of lesions in other parts of the alimentary canal.

This further study showed that injection of the extract is capable of causing a temporary inhibition of the secretion of hydrochloric acid so that the stomach will not react to the histamine stimulus for a period of about six hours.

In addition, profound changes occur in the blood of animals given injections of this extract. A severe anemia develops by the fifth day, and then reticulocytosis appears in which the number of reticulocytes may rise to as high as 50 per cent.

LANGMANN, New York.

HISTOLOGY OF CRANIAL BONES IN EXPERIMENTAL RICKETS AND OSTEOMALACIA IN RABBITS. K. SASAKAWA, J. Pediat. (Tokyo), no. 420, May 20, 1935, p. 598.

Sasakawa produced experimental rickets and osteomalacia in rabbits of varying ages with Steenbock diet 2965 (with an excess of calcium). The osseous changes in rapidly growing animals (30 days old) were similar to those seen in true clinical rickets; in slowly growing animals (60 days old) the picture was that of late rickets (juvenile osteomalacia), and in mature animals (3 years) it was that of atrophic, senile osteomalacia. The author regards all types as different manifestations of the same pathologic process, namely, the disturbance of the calcium-phosphorus metabolism, in animals with different rates of growth. Histologically, he found no osteoid tissue in the cranium of rachitic animals, while in late rickets the osteoid tissue was found only in older animals. In the cranial bones of rabbits with experimental osteomalacia the osteoid tissue was constantly hyperplastic. The author regards the rachitic changes as due to decalcification.

KATO, Chicago.

ACTION OF THE PARATHORMONE (COLLIP) ON BONE TISSUE. ÅKE WILTON, Acta pædiat. **17**:72, 1934.

Between 1930 and 1932 Jaffe, Bodansky and Blair established the fact that parathyroid extract, when administered in large doses to young animals, results in the same kind of disintegration of bone which is seen in osteitis fibrosa generalisata (hyperparathyroidism). These authors, as well as numerous investigators who have followed them, interpret this phenomenon from a predominantly chemical point of view. They assert that the resorption of bone in this case is due primarily to disturbances involving the intermediary metabolism of calcium and of its compounds and that the fibrosis which occurs is to be regarded as a sequel to these alterations. In a series of experiments on young guinea-pigs which are

described in this article Wilton has confirmed the findings of Jaffe, Bodansky and Blair; however, he differs from them diametrically in the interpretation of these findings. Starting from the syncytial doctrine of Hansen and Haggqvist, he analyzes his experimental results from the point of view of cellular pathology rather than from that of chemistry. He concludes that the immediate effect of a surplus of the parathyroid hormone is to produce alterations in the metabolism of the osteocytes, the most important of which is the mobilization of collagen. Liberation of calcium from the skeleton is the result, not the cause, of the cellular changes.

The paper is illustrated by two photomicrographs in color. There is a bibliography of forty references to the pertinent literature.

McCune, New York.

A Simple and Exact Test for Bilirubin in Urine. A. W. J. H. Hoitink, Nederl. tijdschr. v. geneesk. **79**:2928 (June 15) 1935.

The author describes a simple and delicate test for detecting bilirubin in urine (modified Fouchet's test [Godfried: *Biochem. J.* **28**:2056, 1934]). About 10 cc. of urine is put in a test tube. If the reaction is not weakly acid, make it weakly acid (for this purpose use acetic acid or hydrochloric acid). Add half the volume (5 cc.) of a 10 per cent solution of barium chloride. Filter after mixing well. Spread the filter paper with the precipitate on a milk-glass plate or on a piece of white filter paper. Apply 1 drop of Fouchet's reagent in three different places on the border of the precipitate. Wait five minutes. A blue or green color indicates bilirubin. This test is more delicate than that of Gmelin or that of Huppert, Salkowski and Steensma. Five hundredths milligram of bilirubin per liter of urine could be detected.

van Creveld, Amsterdam, Netherlands.

Nutrition

Milk. Horace W. Soper, Am. J. Digest. Dis. & Nutrition **2**:113 (April) 1935.

Soper shows the shortcomings of both raw and pasteurized milk and the relative high efficiency of evaporated milk, so far as cleanliness and digestibility are concerned.

His conclusions are: 1. Raw milk is unfit for human consumption. 2. Pasteurized milk as it reaches the consumer usually contains pathogenic bacteria and is not to be relied on as a safe food. 3. Research into the habits of the people of ancient as well as of modern civilizations reveals that they were consumers of milk, and all suffered from dental caries. 4. Canned evaporated milk is sterile, more easily digested, keeps in the tin indefinitely without refrigeration and contains all the elements of nutrition desirable in milk. Its general use by the public would mean a tremendous economic gain.

Morrison, Savannah, Ga.

Vitamin A Content of Human Liver. Paul D. Crimm and Darwin M. Short, Am. J. M. Sc. **189**:571 (April) 1935.

Spectrophotometric determinations of the vitamin A content in fifteen human livers were made. Seven persons who died accidentally averaged 331 U. S. P. units of vitamin A per gram of liver; two children averaged 80 U. S. P. units per gram, and four patients with pulmonary tuberculosis on a high vitamin diet averaged 523 U. S. P. units per gram. The addition of vitamin A as halibut liver oil results in storage of a large amount of vitamin A in the human liver.

Henske, Omaha.

UNSATURATED FATTY ACID (VITAMIN F) DEFICIENCY. MILDRED ONCKEN, Illinois M. J. **67**:236 (March) 1935.

Oncken points out that carefully executed experiments show that if all neutral fats are excluded from the diet but compensating amounts of fat-soluble vitamins known at present are returned in the form of essentially nonfatty concentrates, animals fail to grow properly and a certain syndrome of symptoms appears. Unsaturated fatty acids completely abolish the symptoms of fat deficiency. If they are contained in the diet they prevent the appearance of the deficiency syndrome.

Blood chemistry determinations have shown that in infectious fevers the blood cholesterol content first decreases and then with convalescence increases, reaching values which may even exceed the normal. At the same time, the iodine number of the fatty material of the blood decreases at the beginning of an infection and increases with convalescence, but very slowly. In infectious diseases the power of the organism to desaturate fatty acids, ascribed largely to the liver, appears to be lowered.

Oncken believes that a closer study of the amount of unsaturated fatty acids in the blood may do much to afford therapeutic control of convalescence from acute infections. Her experience has led her to believe that it may even point the way to the prevention of simpler infections, such as the common cold, and that it is not impossible that the exanthems are correlated with unsaturated fatty acid dyscrasia.

Other observers have demonstrated a striking relation between the deficiency syndrome in animals and that in infantile eczema. Infants suffering from the acute phase of infantile eczema have a lowered unsaturated fatty acid content of the blood. Sources of linoleic and linolenic acids, such as linseed oil, correct the cutaneous condition clinically, while at the same time the unsaturated fatty acid content of the blood increases to a normal level.

The author states that the normal function of the liver is concerned with the desaturation of fats, the better to meet the requirements of the various cells of the body abounding in unsaturated fatty acids and that contemporary food trends, through the introduction on a large scale of vitamin-depleted, fatty acid-extinguished fat supplies, directly violate and oppose the physiologic activities of the body. The liberal feeding of perverted and denatured fat supplies to the children and adults of a nation in place of vegetable oils expressed in natural and unmodified form is of pertinent significance, of which practitioners of medicine may be well aware.

BARBOUR, Peoria, Ill.

THE PROVITAMIN D OF CHOLESTEROL: I. THE ANTIRACHITIC EFFICACY OF IRRADIATED CHOLESTEROL. J. WADDELL, J. Biol. Chem. **105**:771 (July) 1934.

Irradiated cholesterol is more efficacious in preventing rachitic manifestations in chicks than an equivalent number of rat units of irradiated ergosterol; it has an antirachitic value as great as the vitamin D of cod liver oil. Evidence is presented to show that this high potency is due to the activated provitamin previously identified as ergosterol and that it is not due to other substances that may be activated. Pure ergosterol mixed with cholesterol and irradiated does not show enhanced antirachitic value for chicks. It is concluded that the provitamin constituent of cholesterol is a substance different from ergosterol.

MCBRYDE, Durham, N. C.

HUMAN MILK STUDIES: XV. THE NON-PROTEIN NITROGEN CONSTITUENTS. B. N. ERICKSON, M. GULICK, H. A. HUNSCHER and I. G. MACY, J. Biol. Chem. **106**:145 (Aug.) 1934.

The blood and milk of mothers were analyzed for nonprotein, urea and amino-acid nitrogen and for the uric acid content. The data indicate that under normal physiologic conditions the concentration of these constituents is similar in the blood and milk. The nonprotein nitrogen partition of milk in the initial days of

lactation and also during fever is irregular; the nonprotein nitrogen, uric acid, creatine and creatinine are elevated. There was no variation of these constituents in milk in the first and last halves of the nursing period or in milk from the two breasts.

McBryde, Durham, N. C.

The Sparing Action of Fat on Vitamin B: VI. The Influence of the Levels of Protein and Vitamin G. H. M. Evans, S. Lepkovsky and E. A. Murphy, J. Biol. Chem. **107**:429 (Nov.) 1934.

In order to enable the rat to make considerable growth and to survive for a long time on diets free from vitamin B it is necessary to have high levels of fat, protein and vitamin G in the diet. Fat cannot exert its optimal sparing action unless both the protein and the vitamin G content of the diet are high.

McBryde, Durham, N. C.

The Sparing Action of Fat on Vitamin G. H. M. Evans, S. Lepkovsky and E. A. Murphy, J. Biol. Chem. **107**:443 (Nov.) 1934.

The addition of fat exerts no beneficial effect on the growth of rats on diets low in vitamin G and fat.

McBryde, Durham, N. C.

The Changes in the Distribution of Body Water Accompanying Increase and Decrease in Extracellular Electrolyte. Daniel C. Darrow and Herman Yannet, J. Clin. Investigation **14**:266 (March) 1935.

In dogs, rabbits and monkeys intraperitoneal injections of a 5 per cent solution of dextrose and of a 1.8 per cent solution of sodium chloride were followed by an exchange of dextrose, electrolytes and water between the blood and peritoneal fluid until, after from four to six hours, the fluid in the peritoneal cavity was in each case in approximate equilibrium with the blood plasma, and the total volume was approximately that injected. The two solutions thus injected induced, respectively, (1) loss of the extracellular electrolyte, with little change in the total water content of the body, and (2) increase in extracellular electrolyte, with little change in the total water content of the body. The first induced symptoms and signs of dehydration; the second, few symptoms except thirst. The factors governing the intracellular and extracellular distribution of body water are discussed.

Guest, Cincinnati.

The Vitamin B and G Content of Prunes. Agnes F. Morgan and others, J. Nutrition **9**:395 (April) 1935.

This study is a continuance of the previous work done to determine the effect of various methods of drying on the vitamin content of fruit. Rats were used as experimental animals. The results of the experiment indicate that dried California prune flesh contains about 266 Sherman units of vitamin G (B_2) per hundred grams. No other fruit has been reported to contain as much vitamin G. The amount is comparable to that found in wheat germ and is from one-half to one-fourth that of dried milk, eggs, spinach, yeast and liver.

Vitamin B (B_1) was found in the dried prune flesh to the amount of from 80 to 100 Sherman units per hundred grams, or about one-tenth the amount found in wheat germ.

Munns, Winnetka, Ill.

The Effects of Lactose on Growth and Longevity. E. O. Whittier and others, J. Nutrition **9**:521 (April) 1935.

For some time there has been considerable controversy as to the relative advantages of various sugars for use in the infant dietary. Some workers have secured results favoring carbohydrates such as sucrose, maltose and dextrose.

Other competent observers believe that lactose has given superior results. The authors, in conducting a well controlled procedure in which rats and pigs were used as experimental animals, were led to conclude that lactose caused more rapid growth of young rats than did sucrose and that the rats fed lactose in general lived longer than those fed sucrose. They also observed that feeding of excessive quantities of lactose to rats caused diarrhea and retardation of growth. However, each of these reactions was of short duration, and the disadvantage was overcome.

Adult rats fed sucrose ad libitum became heavier than those fed lactose in a like manner. This was due to a greater accumulation of fat. Pigs responded in a similar manner.

Dextrin was found to parallel sucrose in its effect more nearly than it did lactose.

The authors suggest that the results may be directly applicable to human nutrition.

MUNNS, Winnetka, Ill.

GASTRIC SECRETION IN COELIAC DISEASE. J. W. OGILVIE, Arch. Dis. Childhood **10**:93 (April) 1935.

The results of gastric analysis in the series of seventeen cases of celiac disease recorded in this communication confirm the findings of other workers that there is impaired secretion of free hydrochloric acid in the stomach but that true achlorhydria does not occur. There was no diminution in peptic activity, but a slight prolongation was noted in the emptying time. The total chloride content was increased, a finding which is presumably the result of the greater concentration of chloride in the blood, which is to be attributed to the decrease in the red cell count and the increase in plasma. The hypochlorhydria is probably an important etiologic factor in the production of hypochromic anemia in patients with celiac disease.

KELLY, Milwaukee.

BLOOD TRANSFUSION IN THE MALNUTRITION OF INFANCY. M. L. THOMSON, Arch. Dis. Childhood **10**:109 (April) 1935.

Thomson gives the results of transfusions in several malnourished infants and a group of infants with enteritis. In the advanced stage of acute enteritis blood transfusions should be a routine measure. A favorable response can be expected only in a small proportion of such cases. In the marasmic infant blood transfusion should be employed as soon as the condition of acute enteritis supervenes. In uncomplicated cases of malnutrition blood transfusion is a valuable measure. A favorable response may be expected in a large percentage of such cases.

KELLY, Milwaukee.

THE RED CORPUSCLES IN ACIDOSIS AND ALKALOSIS. W. T. W. PAXTON, Arch. Dis. Childhood **10**:115 (April) 1935.

In acidosis, whether occurring in disease or induced by drugs, the red corpuscles are enlarged, the cell apparently retaining its normal shape. This may be accompanied by an increase in the red cell count, which, when it occurs, is probably due to concentration of blood. Alkalosis causes a reduction in the size of the corpuscles, without change in the shape or the hemoglobin content. There is probably no change in the red cell count due to alkalosis per se. The red corpuscles in early infancy, in spite of their different features, react to alkalosis in the same way as those of the older person.

KELLY, Milwaukee.

TIME FACTORS IN THE PASTEURIZATION OF MILK. I. WALKER HALL, Brit. M. J. **1**:972 (May 11) 1935.

Hall discusses the methods of pasteurization, with especial reference to "flash" heating, and pleads for improvement of the methods of pasteurization in general for the protection of the public.

ROYSTER, University, Va.

"Digestibility" of Common Foodstuffs as Determined by Radiography. W. C. D. Maile and K. J. L. Scott, Lancet **1**:21 (Jan. 5) 1935.

The authors refer to the extensive investigations on "gastric response" to various foods carried out by Rehfuss, Hawk and their associates from 1918 to 1920 with the use of the Rehfuss tube.

In the present investigation eight subjects were studied. Test meals were taken in the morning when the stomach was assumed to be empty. The barium sulfate was either mixed with the food or taken with a little water during the eating of solid food. The amount of barium sulfate administered was usually an ounce (28.35 Gm.).

The following conclusions were arrived at: (1) An ordinary meal leaves the stomach in about four hours; a very large meal may be retained for five hours. (2) A meal not necessarily large but containing much butter or cream is retained in the stomach for a longer period. (3) Concentrated carbohydrates, such as sugar, leave the stomach much more quickly than natural carbohydrates, such as banana or potato. (4) Cooking shortens the time of gastric digestion for some foods and increases it for others. (5) The length of time required for digestion of different foods in the stomach cannot be taken as a measure of their "digestibility." (6) Fat in abnormal proportions causes the emptying time of the stomach to be prolonged to a marked degree. (7) The sensation of hunger does not appear to be dependent on vigorous peristalsis.

Langmann, New York.

So-Called Acidosis Attacks: A Plea for More Accurate Diagnosis. Donald Paterson, Lancet **1**:917 (April 20) 1935.

The factor of infection is stressed in the so-called attacks of acidosis, as in the majority of children acidosis or ketosis by itself leads to little or no change in the clinical picture.

Most authorities agree that cyclic vomiting is a metabolic disturbance; they cannot decide whether this disturbance is due to infective, dietetic, psychologic or allergic factors. It is Paterson's belief that too little stress has been laid on infection as the cause of these attacks.

During the actual attack fats should be vigorously excluded from the diet, but once the attack is over the child should be put back on a normal diet. In the treatment of an acute attack careful examination is necessary to exclude organic disease.

The removal of foci of infection, such as diseased tonsils, is an important measure in the prevention of attacks. The mother of the child prone to these attacks may do much to forestall an attack by watching the stools daily; when they become pale she can reduce the fat temporarily and administer dextrose and a mild saline aperient.

Langmann, New York.

Annual Cycle in the Secretion of Vitamin C by the Mammary Gland of Milch Cows. P. Rohmer, N. Bezssonoff and E. Stoerr, Bull. Soc. de pédiat. de Paris **32**:566 (Nov.) 1934.

Three different batches of milk were tested daily for the vitamin C content, one from a Strasbourg dairy, a second from a farm and a third from the cows of a village peasant. The curves for the year ran practically parallel for the three milks. The amount of vitamin C in the milk does not vary directly with the variation in the amount contained in the intake of food of the cows. The animals can synthesize the vitamin independently of its presence in their food. In studies on infants it was found that they are able to synthesize vitamin C. This ability is most marked in the first six months of life, when the infant uses up about four times the amount of vitamin C that is supplied to it in breast milk; it diminishes during the second half of the first year and is apparently completely lost by the beginning of the second year. It was found that one of the important factors required by the infant for the synthesis of vitamin C is vitamin A; when the latter

is removed from the diet the former ceases to be manufactured, and when vitamin A is restored in the diet vitamin C appears in the body, even though the diet is kept free from vitamin C.

BENJAMIN, Montreal, Canada.

INFLUENCE OF MASSIVE DOSES OF VITAMIN A ON ANIMALS DEPRIVED OF SAME IN THE DIET. A. CHEVALLIER, L. CORNIL and P. CHABRE, Compt. rend. Soc. de biol. **115**:541 (Jan. 9) 1934.

Experiment on Rats.—Two rats used as controls were fed a diet devoid of vitamin A and were permitted to go on to death. Two others received 0.025 mg. of vitamin A daily, with subsequent normal growth, gaining about 5 Gm. per week. Others on a diet without vitamin A were given 80 mg. daily, which was followed by death and signs comparable to avitaminosis A in about three weeks. The animals showed: (*a*) marked loss of weight and complete anorexia; (*b*) dry, reddened corneas with concretions and diminution of the palpebral fissures, and (*c*) important changes in the skin, such as desquamation, bristly fur and falling hair.

Experiment with Guinea-Pigs.—Six guinea-pigs were fed bread, bran and wheat but no green vegetables; at the same time they received 400 mg. of vitamin A daily. The weight soon decreased from 500 to 300 Gm., with subsequent death in from fifteen to sixteen days; there were intestinal hemorrhages, loss of hair in tufts and marked anorexia ante mortem.

Conclusion.—Rats and guinea-pigs on diets devoid of vitamin A fed massive doses of vitamin A succumb and show symptoms and signs similar to those produced by avitaminosis A.

WAHL, Cleveland.

RICKETS. A. B. MARFAN, Nourrisson **23**:73 (March) 1935.

Marfan believes that vitamin D deficiency is not the fundamental cause of rickets, but that the underlying cause is some infection or intoxication. He mentions as possible agents congenital syphilis, tuberculosis, bronchopneumonia, pyodermas, chronic diphtheria and malaria. Digestive disturbances such as chronic diarrhea or celiac disease may be responsible, or such chronic diseases as nephritis, diabetes and scurvy. These diseases may directly cause a decrease in the vitamin D factor, or the decrease may be due to other recognized factors such as lack of sunshine or lack of ultraviolet rays from other sources. Even with a lowering of the content of vitamin D, which is contained in the activated ergosterol of the body, Marfan does not believe that rickets develops unless the chronic infection or intoxication acts on the bone-developing areas.

MCCLELLAND, Cleveland.

EVAPORATED MILK FOR INFANT FEEDING. A. B. MARFAN and G. BLECHMANN, Nourrisson **23**:99 (March) 1935.

In the case of infants with malnutrition or hypothrepsia the authors believe that an evaporated milk with half of the cream removed and some carbohydrate added gives better results than the plain evaporated milk.

MCCLELLAND, Cleveland.

CAUSES OF THE DIFFERENT TYPES OF RICKETS. A. B. MARFAN, Nourrisson **23**: 137 (May) 1935.

Marfan believes that rickets at a given age affects the bones which are at that time the seat of most active osteogenesis. Thus, rickets appearing during the first few months affects the cranial bones; later on in the first year the ribs and bones of the upper extremities suffer, while after the first or second year the bones of the lower extremities are involved.

The direct cause of the rickets is the depletion of the antirachitic factor in the body. This may be due to an inadequate intake of the antirachitic factor or to excessive destruction.

The destructive influences vary at different ages. Thus, the most frequent cause of rachitic craniotabes is congenital syphilis. Other less frequent causes of this early type are chronic nasal diphtheria, chronic pyodermas, and eclampsia in the mother.

During the latter half of the first year rickets is usually caused by improper alimentation or by chronic diarrhea. Other causes at this age are scurvy, tuberculosis and subacute bronchopneumonia.

The late type of rickets is most often attributable to chronic renal disease. Inadequately treated congenital syphilis or chronic indigestion as in celiac disease may also be a cause.

The extent of the deformities caused by rickets depends on the duration of action of the infection or intoxication on the antirachitic factor. Anhelation or the ingestion of inadequate amounts of antirachitic factor may be contributory factors in the failure of osteophylaxis. McCLELLAND, Cleveland.

CHINESE METHOD OF ACUPUNCTURE IN THE TREATMENT OF NURSLINGS WITH ANOREXIA. M. LAVERGNE, Nourrisson **23**:154 (May) 1935.

The Chinese method of acupuncture was practiced on twenty-eight infants with functional anorexia. The site of puncture was the anterior surface of the fibula, 3 cm. above the external malleolus. The results were poor or mediocre in ten cases but good or excellent in the remaining eighteen. McCLELLAND, Cleveland.

THE STAGES OF CURABILITY AND INCURABILITY IN ALIMENTARY DYSTROPHIES. G. MOURIQUAND, Presse méd. **43**:489 (March 27) 1935.

A state of general dystrophy in the infant may be brought about by athrepsia of alimentary origin. A diet deficient in vitamins leads to an athrepsia which is curable in the earlier stages and, later, to "functional death," announced by a sudden and rapid loss of weight, lowering of the basal metabolic rate and impairment of digestive power, as manifested by a dystrophic diarrhea. This terminal diarrhea heralds the failure of all therapeutic measures. Mouriquand divides the phases of deficiency dystrophy, with particular reference to vitamin C deficiency, into two general classes: (1) the eutrophic stage and (2) the dystrophic stage. The eutrophic stage may in turn be divided into stages of predeficiency, with no discernible changes; inapparent dystrophy, when changes may be determined by roentgenography, laboratory examinations, electrical reactions and other means, and the stage of actual deficiency, manifested by osseous and muscular changes, anemia, gingivitis and other signs. Between the eutrophic and dystrophic stages there exists, in some forms of deficiency at least, an intermediate stage of some days when there is cessation of growth, with marked signs of deficiency and failure of the glands of internal secretion to function normally.

The dystrophic phase is marked by failure of assimilation and may be divided into a stage of curability and the terminal stage of incurable deficiency and general dystrophy, with diarrhea and complete breakdown of intestinal function.

BIEKER, St. Petersburg, Fla.

RÔLE OF BLOOD CALCIUM. A. VALATX, Progrès méd., April 6, 1935, p. 570.

This article offers a brief review of studies on the blood calcium content, emphasizing the importance not only of changes in the total blood calcium content but also in the calcium partition. Methods of determining the latter are difficult, and even in estimating the serum calcium content errors of from 3 to 5 per cent may occur. Further, the calcium content of serum or whole blood does not always give definite information regarding the calcium content of other tissues. An excellent bibliography is included. LESLIE, Evanston, Ill.

PELLAGRA AND "UNAPPARENT DYSTROPHY" IN THE CHILD. M. PAVIA, Riv. di clin. pediat. **33**:397 (April) 1935.

The provincial health department of Verona reported twenty-eight cases of pellagra in the seven and one-half years up to June 1934. Most of these cases were in adults. The author had an opportunity to observe three children with pellagra. A review of the history of the prepellagrous period and examination of the siblings of one of the patients led Pavia to believe that hidden pellagra ("unapparent dystrophy") is possible and not rare in children.

HIGGINS, Boston.

THE AUTONOMIC SYSTEM AND EPINEPHRINE: THE INFLUENCE OF EPINEPHRINE ON THE AMOUNT OF WATER IN THE BLOOD. A. FIANO, Riv. di clin. pediat. **33**:513 (May) 1935.

Observations were made on nine fasting infants between 7 and 12 months of age. On one day 1 cc. of epinephrine hydrochloride was injected. On another day controlled observations were made. The hemoglobin content, red cell count and albumin content of the blood were observed for a period of four hours.

With but one exception, the albumin content was found to be elevated for one-half hour after the injection of epinephrine. The red cell count and hemoglobin content were unchanged. This indicated a loss of water from the blood.

Under normal conditions during fasting the amount of water in the blood is not constant, but small oscillations occur.

HIGGINS, Boston.

ALKALI RESERVE AND INORGANIC PHOSPHORUS CONTENT OF THE BLOOD IN HEALTHY CHILDREN AND IN CHILDREN WITH VARIOUS INFECTIONS OF THE RESPIRATORY TRACT. A. SALVADEI, Riv. di clin. pediat. **33**:566 (May) 1935.

Observations were made on thirty-one well children and on sixty-one children with respiratory infections. Children with pneumonia showed a diminution in both the alkali reserve and the inorganic phosphorus content of the blood. Children with acute bronchitis had no changes in the alkali reserve and a slight diminution in the inorganic phosphorus content. The mean findings are given in the following table:

	Alkali Reserve, per Cent by Volume	Inorganic Phosphorus Content, Mg.
Normal	54.5	3.44
Pneumonia	44.1	2.08
Acute bronchitis	53.5	3.04

In patients with chronic respiratory infections (tracheobronchial adenopathy, chronic bronchitis, tuberculous bronchopneumonia, tuberculous pleurisy), the alkali reserve was essentially the same as in normal children, but the inorganic phosphorus content showed some diminution, especially in the tuberculous patients (2.75 mg.).

The maximum diminution in the alkali reserve and in the inorganic phosphorus content of the blood is in the early acute stages of pneumonia. As the disease tends to resolve and the child's condition approaches normal the alkali reserve quickly returns to a normal figure, while the inorganic phosphorus content shows a more gradual return.

HIGGINS, Boston.

ALIMENTARY FEVER. RIETSCHEL, Ergebn. d. inn. Med. u. Kinderh. **47**:185, 1934.

Finkelstein, who created the conception of "alimentary fever", demonstrated clinically that in infants who were given feeding mixtures rich in sugar or to whom sugar or salt was administered parenterally, fever developed in spite of the fact that bacterial action was excluded as a possible etiologic agent. It must be stated, however, that the "sugar fever" as well as the "salt fever" phenomenon could be observed only in dyspeptic or nutritionally disturbed infants. Although at first Finkelstein and his school denied that proteins have any etiologic rôle in

the "fever," later they admitted that albumin may, like sugar and salt, act as a producer of fever. Finkelstein, like Heim and John, stresses loss of fluids from the tissues as a principal cause of the fever but maintains, nevertheless, that the rise in temperature is not a simple physical phenomenon but that it represents a true febrile reaction.

Rietschel and his school explain the alimentary fever as follows: When salt or sugar is administered in concentrated form, the body, in order to restore the osmotic equilibrium in the tissues, must mobilize water in order to transfer the salt or sugar to the blood and tissues and eventually eliminate them through the kidneys. Mobilization of water implies its liberation from the colloids in the subcutaneous tissues. This represents considerable cellular activity, in the course of which heat is liberated. It is clear that the more concentrated the solutions of salt and sugar, the greater the amount of work done by the cells. This liberation of the water is, of course, centrally regulated (water center). However, all the intestinal glands are simultaneously attempting intensive activity to thin down or dilute the salt or sugar in order to obviate the danger of thirst, and in this effort all the body tissues are assisting. As regards the therapy of alimentary fever, two requirements must be fulfilled: first, water must be administered and, second, those substances which led to the exsiccation must be eliminated. Rietschel looks on the transitory fever of the new-born "as of similar pathogenesis" as "alimentary fever" and recommends the same type of therapy, namely, the administration of sufficient fluids, particularly water.

CAPPER, Philadelphia.

HISTOLOGIC INVESTIGATION OF PANCREAS IN NUTRITIONAL DISTURBANCES IN INFANTS. S. MAHRBURG, Pedjatrja polska **14**:179, 1934.

After an extensive study of the literature Mahrburg describes the physiology of the pancreas in infants. On postmortem examination the pancreas was removed from infants who had died of gastro-intestinal disturbances. Various stains were used so as not to alter the picture shown by the tissue. Mahrburg came to the conclusion that any histologic or physiologic pathologic changes of the pancreas were usually due to a condition of the gastro-intestinal tract. However, he never found in his experiments an actual inflammatory process of the pancreas itself.

KRUPINSKI, Chicago.

RICKETS AND SPASMOPHILIA: I. STURE SIWE, Acta pædiat. **17**:1, 1934.

This is the first of a series of three papers based on analysis of the records of 142 cases of infantile tetany, of which 131 were cases of active tetany, and 69, cases of rickets uncomplicated by spasmophilia. The study was commenced in 1932; since that time all patients entering Af Klercker's Clinic in Lund with rickets and convulsive disorders have been studied thoroughly by chemical, electrical and roentgenographic means, in addition to the usual clinical methods. In this introductory paper Siwe discusses a number of loosely related topics bearing on various aspects of tetany.

In regard to seasonal incidence, tetany possesses certain features which suggest that it is somewhat independent of rickets. This suspicion is supported by the fact that a relatively large number of infants show unequivocal tetany during the first six months of life, a period during which demonstrable rickets is uncommon. Tetany is more frequent in boys, whereas the incidence of rickets is equal in both sexes. The reasons for this disparity are uncertain.

One of the most striking features of tetany is the variability of its clinical manifestations. Diminished concentration of calcium in the blood serum is the most important diagnostic criterion; unless measurement of the calcium content is made many cases must necessarily pass unrecognized. The physician who lacks the facilities of a laboratory is justified in employing calcium therapy in the case of any infant who has convulsions at that age and during those seasons of the year when tetany is to be expected. At the time of the first examination of an infant

with active tetany, any or all of the classic triad of symptoms, viz., laryngismus, tetany and convulsions, may be absent. This was true in 10 per cent of the cases in the author's series. The younger the patient, the less characteristic and complete was the symptomatology. In the 131 infants with active tetany symptoms or signs were detected at the first examination according to the following order of frequency: positive electric reactions, 79.1 per cent; convulsions, 77.9 per cent; Chvostek's sign, 66.9 per cent; a positive peroneal reflex, 58.5 per cent; laryngospasm, 54.2 per cent; Trousseau's sign, 38.2 per cent; carpopedal spasm, 17.6 per cent. Although prolonged clinical observation may clarify the diagnosis in doubtful cases, it is unwise to dispense with the determination of the serum calcium content. Unfortunately the concentration of calcium will not always be found to be diminished at the first examination; there is a surprising lack of parallelism between the level of calcium in the serum and the severity of the clinical manifestations. Siwe presents evidence to show that the convulsive seizure itself has a tendency to elevate the total calcium level of the serum. Finally, he suggests that the fever which is present in some cases of tetany may be related more closely to the actual convulsions than to the infectious process which caused them.

Thirty pages are devoted to a tabular synopsis of the cases which form the basis of this paper and of those which follow. McCUNE, New York.

RICKETS AND SPASMOPHILIA: II. THE CHANGES IN THE SPINAL FLUID IN TETANY. STURE SIWE, Acta pædiat. **17**:157, 1934.

The diagnosis of tetany can usually be made with reasonable certainty if the opportunity to observe the patient is sufficiently long and provided that all the accessory methods for diagnosis are available. Occasionally the differentiation of spasmophilia from other causes of convulsions presents great difficulties; this is particularly true when pathologic changes are present in the cerebrospinal fluid. For several years lumbar puncture has been performed as a routine on infants with tetany who have been treated in Af Klercker's clinic in Lund. Usually neither pleocytosis nor an increased protein content is found in the spinal fluid of infants with tetany. Occasionally, however, both the protein content and the number of cells may be slightly increased. Siwe presents synopses of the histories of eight patients to illustrate the confusion in the diagnosis which these findings may produce. He suggests that the circulatory alterations which accompany spasmophilic convulsions are responsible for the pathologic changes in the spinal fluid; a similar phenomenon is thought to occur in epilepsy and in pertussis.

Contrary to the general opinion, the variations of the calcium content of the cerebrospinal fluid of infants with tetany do not parallel the simultaneous fluctuations of the calcium content of the blood serum. Although the number of pertinent observations is too small to be conclusive, Siwe's findings suggest an independent variability of the calcium content in the two mediums. They show that there is a tendency for the calcium content to be higher in the spinal fluid of infants with active tetany than in the spinal fluid of those with latent tetany.

The second portion of this paper is devoted to a discussion of those cases of tetany in which the concentration of calcium in the blood serum is normal. Spasmophilia is an exceedingly variable disease. It has been shown repeatedly that there is a marked lack of proportion between the degree of nervous irritability and the level of the serum calcium. Is it possible that tetany may occur in association with entirely normal values for calcium in the blood serum? Siwe answers this question emphatically in the affirmative, adducing seven cases to fortify his assertion. In most of these cases, if not in all, one or more of the signs of hyperirritability usually ascribed to tetany were present; in all seven cases the concentration of calcium in the serum was normal, at least at the time when blood was drawn for the determinations.

Turning to the consideration of tetany in breast-fed infants, the author reviews the factors which are ordinarily discussed in regard to the pathogenesis of this condition, namely, an inadequate intake of calcium, lack of vitamin D and deficient

function of the parathyroid glands, hypothetically resulting from injury. Siwe's series of patients included three breast-fed infants with tetany. In each case the concentration of calcium in the mother's milk was lower than normal, that is, approximately 25 mg. per hundred cubic centimeters instead of from 34 to 54 mg. per hundred cubic centimeters, which was the mean value that the author obtained by analyzing twenty-three samples of breast milk from eight mothers of normal infants. The symptoms of tetany disappeared promptly when calcium, together with a potent preparation of vitamin D, was added to the diet of the three infants. Siwe asserts that an an inadequate amount of calcium in the diet is one of the important factors that contribute to the occurrence of tetany in breast-fed infants.

McCUNE, New York.

RICKETS AND SPASMOPHILIA: III. RICKETS WITH AND WITHOUT SYMPTOMS OF SPASMOPHILIA. STURE SIWE, Acta pædiat. **17**:184, 1934.

Siwe considers the problem of whether the frequent coincidence of rickets and tetany in the same infant really indicates a close causal relationship between the two, as is universally taught, or whether a more careful scrutiny of evidence does not suggest that rickets and tetany are pathogenetically independent diseases the similar etiology of which is attested by the fact that both can be treated successfully by the same methods.

Coincidence furnishes the strongest proof that they are closely related, yet it is superfluous to state that infants who have rickets do not always have tetany; furthermore, Siwe's series includes a number of cases of tetany in which not the slightest sign of rickets was revealed by thorough application of the most modern methods of examination. The author admits freely that the disturbance of metabolism which is fundamental to rickets might have transpired before the time of examination; in any event, it had left no residual traces. In the second place, if "tetany is a complication of rickets" the curve for the seasonal occurrence of spasmophilia should lag somewhat behind the curve for the seasonal occurrence of rickets. Actually those portions of the two curves which represent the greatest number of cases nearly coincide; both rickets and tetany occur with greatest frequency from the first of December through March; from April to July there are more cases of rickets without tetany than with it, although the natural tendency for rickets to heal is more marked during the summer than in the winter months. A third argument for the pathogenetic independence of the two diseases is the fact that the annual incidence of each is not the same; hence the terms "rickets year" and "tetany year;" these years do not coincide. To explain these facts, which are inconsistent with the unitarian theory of pathogenesis, German and Austrian authors have assumed "an individual predisposition to tetany." No one has dared previously to suggest that rickets and tetany may be pathogenetically independent.

A further fact which requires verification and explanation is the probability that tetany occurs more frequently in infants under 4 months of age than does rickets.

The greatest obstacle to the solution of the foregoing implied problems is the difficulty of determining exactly both the presence of rickets and the degree of its activity in a given case. Roentgenographic study is superior to physical examination in this respect, although changes shown by the roentgen rays lag behind chemical alterations in the blood serum and do not indicate accurately either whether the disease is active at the time of examination or, in some cases, whether it is present. A single determination of the concentration of calcium and phosphorus in the serum is not to be depended on to establish a diagnosis of the presence of rickets or of its activity. The time during which the rachitogenic disturbance of metabolism has been present is of great importance in conditioning the chemical findings, the roentgenographic features and the chemical composition of the serum.

Siwe does not agree that lowering of the concentration of inorganic phosphorus in the serum is the first happening in the evolution of rickets; this is

probably preceded by an aberration of metabolism which is of more fundamental moment. Furthermore, he failed to find a reciprocal relationship between the serum calcium content and the serum phosphorous content; in some cases the fluctuations of the two are parallel. Whether rickets heals rapidly or slowly, he did not find that a rise in the concentration of phosphorus is accompanied by a fall in the calcium content. This makes it questionable whether the activation of tetany which is seen frequently at the beginning of treatment of rickets with vitamin D or ultraviolet rays is due to decrease of the concentration of calcium in the serum.

Siwe agrees with Shipley and Park and with Block that the differentiation of rickets into two types (high calcium rickets and low calcium rickets) is justified. Although in the majority of cases rickets is characterized by a decreased concentration of phosphorus in the serum, there is a smaller group of cases in which the calcium content is relatively lower than the phosphorus content. Most authors have stressed the almost invariable occurrence of tetany in this type of case. This emphasis weakens the validity of the second category by suggesting that low calcium rickets represents simply ordinary rickets which shows the chemical changes of impending tetany. Siwe reports ten cases of active rickets associated with marked lowering of the serum calcium content in which, although the calcium content remained for some time at the level at which it is noted in tetany, no symptoms of spasmophilia were manifest.

In the second part of the paper the author discusses the relation between the symptoms of rickets and the chemical composition of the blood. Craniotabes is the principal osseous sign in cases of low calcium rickets; tetany is no more frequent in these cases than in patients without craniotabes. In patients in whom rickets led to pathologic fractures the serum calcium content was found to be slightly higher than in patients without fractures. Patients who exhibited hypotonia showed no marked difference in the chemical findings of the blood as compared with other patients with rickets but with normal muscular tonus.

The series of papers is concluded with a recapitulation, and a bibliography of thirty-five references to the English, American, French, German and Scandinavian literatures.

McCune, New York.

Prenatal Conditions

Congenital Absence of the Sacrum. W. R. Hamsa, Arch. Surg. **30**:657 (April) 1935.

Hamsa adds two cases to the reported eighteen cases of this deformity. This deformity is usually associated with others, but there are some cases in which the degree of the deformity is compatible with life. The manifestations of the deformity itself are a characteristic flattening of the contour of the caudal region, absence of the buttock prominences and the groove between them, absence of sacral spinous processes and on rectal examination the encountering of soft tissue instead of the normal bony concavity of the sacrococcygeal segments. The roentgenologic findings are characteristic. With the typical neurologic and orthopedic manifestations are primarily associated complete incontinence of urine and, occasionally, incontinence of feces as well. Treatment is directed toward correction of the deformity of the lower extremity if sufficiently active muscular action is present to indicate that the patient will be able to assume the erect position when the lower extremities and the spine are alined.

Kaiser, Rochester, N. Y.

Congenital Heart Disease. D. C. Muir and J. W. Brown, Brit. M. J. **1**:966 (May 11) 1935.

Muir and Brown discuss the signs and symptoms of the various forms of congenital defects of the heart.

Royster, University, Va.

SPECIMENS FROM A CASE OF CONGENITAL OBSTRUCTION OF THE ESOPHAGUS. HAZEL CHODAK GREGORY, Proc. Roy. Soc. Med. **28**:837 (May) 1935.

This case was reported on Oct. 26, 1934. Pathologic specimens of the stomach and esophagus with microscopic sections were shown. The subsequent history and a full report of the autopsy are given. WILLIAMSON, New Orleans.

SPECIMENS FROM A CASE OF RIGHT-SIDED HEMIHYPERTROPHY ASSOCIATED WITH PUBERTAS PRAECOX. JOAN HARWOOD and ELIZABETH O'FLYNN, Proc. Roy. Soc. Med. **28**:837 (May) 1935.

The patient from whom the specimens were shown was a boy of 6½ years, whose case was described in the *Proceedings* (**25**:957, 1932). The authors give a summary of this previous report and present the later findings and the report on the autopsy performed thirty-six hours after death.

WILLIAMSON, New Orleans.

SPECIMENS FROM A CASE OF CONGENITAL HYPERPLASIA OF THE SUPRARENAL CORTEX. J. VERNON BRAITHWAITE, Proc. Roy. Soc. Med. **28**:839 (May) 1935.

The patient was shown at the meeting of the Section for the Study of Disease in Children held in November 1934, and the case was reported under the title "Giantism, Virilism, and Pseudohermaphroditism." The operation (adrenalectomy on the left side) and the observations at postmortem examination are described in detail in the report. WILLIAMSON, New Orleans.

A CASE OF IMPERFORATE ANUS. E. IRRMANN and P. MULLER, Bull. Soc. d'obst. et de gynéc. **24**:301 (April) 1935.

The infant was normally delivered at full term and weighed 3,000 Gm. at birth. An imperforate anus with complete absence of the radial anal folds and pigmentation was noted. No rectal bulge could be felt when the infant cried. As it was believed that this was a case of rectal atresia as well as imperforate anus, sigmoidostomy was made on the left, with the area under local anesthesia. The artificial anus functioned well, and the child was discharged from the hospital, but returned in three months. Irrmann and Muller discuss the problems which had to be decided at that time. These involved a study of the degree of atresia, on which depended the possibility of establishing an artificial perineal anus.

GLASER, Rochester, N. Y.

CONGENITAL ABSENCE OF THE APPENDIX. J. LOUYOT, J. RICHON and LACOURT, Nourrisson **23**:101 (March) 1935.

Another case of this rare congenital anomaly is reported. In regard to the etiology, syphilis was suspected, though positive evidence was not obtained at necropsy. The baby died from a cerebral hemorrhage.

MCCLELLAND, Cleveland.

A CASE OF CONGENITAL MITRAL MALFORMATION. D. ROUTIER and G. POUMEAU-DELILLE, Paris méd. **1**:416 (May 4) 1935.

A man died when 36 years of age from cardiac decompensation. Autopsy revealed an enormously dilated left auricle from which a small aorta arose. The mitral valves did not permit closure and the left ventricle was a mere appendage.

LESLIE, Evanston, Ill.

CONGENITAL ABSENCE OF THE FIBULA. LAPASSET and CAHUZAC, Rev. d'orthop. **22**:110 (March) 1935.

Congenital absence of the fibula is not uncommon. Rabaud and Hovelacque have reported seven cases in seventeen members of one family, and Hovelacque

and Noël have shown that the condition may occur in several generations, so that a familial history is a factor. Sex is not a predisposing factor. The fibula may be absent in toto or partially, and such congenital deformity is rarely isolated but is usually accompanied by other manifestations of defects. The opposite fibula is frequently also deformed; the tibia may be shortened and bent and may be incomplete, with usually the upper epiphysis absent, the internal malleolus increased in size, an exaggerated normal curve of the bone and an atrophic external condyle. The patella may be normal, atrophic or absent. There may be malformation of the foot, with fusion of the tarsals and absence of the astragalus, cuboid or the second and third cuneiform bone. There may be supernumerary toes. Accompanying the lesions there are usually some muscles absent, and dislocation of the hip, genu valgum, hernias, ectopic testicles and poor teeth may also be part of the picture. The condition is developmental in origin. Treatment consists in maintaining as good corrected position of the deformed leg as possible, with appropriate surgical measures to correct the deformity. RYAN, Vancouver, Canada.

PARTIAL CONGENITAL ABSENCE OF THE INTESTINE. ALDO BOLLETTINO, Pediatria **43**:192 (Feb.) 1935.

A case of partial congenital absence of the intestine is reported, with a detailed discussion of the clinical and anatomicopathologic features. Interesting pictures and photomicrographs of the lesions add to the value of the presentation.

SIGNORELLI, New Orleans.

Diseases of the New-Born

THE NEWBORN INFANT. JESSE R. GERSTLEY, M. Clin. North America **18**:1403 (March) 1935.

The various conditions encountered in and the peculiar problems met with during the neonatal period are covered in this paper. The subjects of injuries at birth, congenital abnormalities, infections and nutrition are discussed in some detail, with special emphasis on the rational management of these children.

EUGENE SMITH, Ogden, Utah.

OSTEOMYELITIS OF THE MAXILLA IN THE NEWLY-BORN. H. D. WHITE, Arch. Dis. Childhood **10**:85 (April) 1935.

Osteomyelitis of the maxilla in the new-born is a bacterial infection, usually staphylococcic or streptococcic, affecting healthy children between the ages of 2 weeks and 9 months. It is a hematogenous infection localizing in the maxilla, either in the bone itself, in the region of the center of ossification or in a tooth bud. The pathologic changes are the same as those in osteomyelitis in older children. The symptoms—fever, swelling of the face and eyelids, discharge from the nostril and swelling and discharge from the alveolar margin—are clearcut and easily recognized. Some operative measure is indicated in the majority of cases and should be instituted early. The prognosis is first serious, but if improvement follows operation complete recovery is usual. KELLY, Milwaukee.

A CASE OF TWINS IN WHICH THE SECOND FOETUS PRESENTED BY THE THORAX. H. E. RODWAY, J. Obst. & Gynæc. Brit. Emp. **42**:304 (April) 1935.

Rodway presents a case involving a 22 year old primipara with normal pelvic measurements. Roentgenograms taken three weeks before delivery showed that two fetuses were presenting by the vertex. The prenatal course was uneventful.

Following spontaneous rupture of the membranes the patient went into labor and in forty-three hours had a spontaneous delivery of a male infant. On further

examination the head of the remaining fetus was found to be in the right iliac fossa; the limbs were at the fundus, and a hand was presenting. After rupture of the membranes it was found that the thorax was impacted in the pelvis, the fetus being folded almost double.

With the patient under open ether anesthesia, the thorax was dislodged, and since the head was out of reach podalic version and extraction were accomplished after the legs were brought down with great difficulty.

The infant was easily revived but sustained a displacement of the epiphysis of the lower end of the femur, which was manipulated by an orthopedic surgeon, and the infant was finally discharged with a normally functioning limb.

ADAIR and UDESKY, Chicago.

RESPIRATORY FAILURE INCLUDING SO-CALLED ASPHYXIA NEONATORUM (GOULSTONIAN LECTURE). ALAN MONCRIEFF, Lancet **1**:531 (March 9); 595 (March 16); 664 (March 23) 1935.

After discussing the causes of respiratory failure in general, Moncrieff devotes a large part of this article to a discussion of the factors concerned in respiratory failure occurring in the neonatal period. The causes of respiratory failure in the new-born are listed as follows:

A. Central

1. Immaturity of respiration center
2. Damage to center, increased intracranial pressure, edema, hemorrhage
3. Narcotics
4. Chemical factors, lack of oxygen, excess of carbon dioxide, etc.
5. Circulatory disturbances affecting center in utero, pressure on cord, etc.

B. Peripheral

1. Obstruction, premature inspiration
2. Delayed expansion
3. Muscular feebleness
4. Circulatory failure

The diagnosis of the type, the cause of respiratory failure in the new-born, and, finally, the treatment are discussed at great length.

In treating respiratory failure in the new-born there is no essential difference between the blue and the white type of asphyxia. Two primary considerations must never be ignored, namely, the supply of warmth and the great importance of gentleness. Clearing out obstructive material in the pharynx by suction often proves effective. The administration of a 5 per cent dilution of carbon dioxide in oxygen should be employed when the first maneuver is not successful in inducing respiration. Direct laryngoscopy, with adequate suction and cleansing of the lower respiratory tract, followed by insufflation of oxygen and carbon dioxide may be necessary in the cases of babies who do not respond to the simpler measures.

LANGMANN, New York.

DISEASES OF THE NEWLY BORN. NORMAN B. CAPON, Practitioner **134**:403 (April) 1935.

The author points out that the neonatal death rate in England and Wales is about 20,000 yearly, and it accounts for almost half the infantile death rate. The chief causes of neonatal illness are discussed at length, i. e., prematurity, asphyxia and trauma at birth. Particular attention is given to the question of the care and feeding of the premature infant. Other conditions discussed include icterus gravis, hemorrhagic disease of the new-born and congenital malformations.

SAPPINGTON, San Francisco.

BIOMETRIC DATA ON THE NORMAL NEAPOLITAN NEW-BORN. E. TATAFIORE, Pediatria **43**:422 (April) 1935.

From the anthropometric study of four hundred and seventy normal new-born in the province of Naples and a comparison of these data with those of other regions of Italy, Tatafiore prophesies improvement for the constitutional development of the Neapolitan of the future. This is attributed to improved hygienic and prophylactic conditions provided by the government.

SIGNORELLI, New Orleans.

CAUSES OF DEATH IN THE NEW-BORN. CARL JOSEPH FISCHER, Wien. klin. Wchnschr. **48**:360 (March 22) 1935.

Fischer reports on the causes of death in six hundred and sixty-nine new-born children. Most of the cases are from the Women's Clinic in Gratz, and some are from St. Anna Children's Hospital of Vienna. All the new-born children born from 1923 to 1932 who died between the first and tenth day of life are considered. Autopsies were performed on all the children, including microscopic examination in cases in which it was thought necessary. The following table shows the causes of death:

Cause	Number	Percentage
1. Intra-uterine death of fetus	115	17.5
2. Craniotomized children	33	5.0
3. Congenital syphilis	50	7.6
4. Congenital malformations	39	5.9
5. General weak condition	39	5.9
6. Traumas at birth	224	34.1
7. Aspiration of amniotic fluid	37	5.6
8. Bronchitis and bronchopneumonia	85	12.9
9. Other causes	36	5.5

ABT, Chicago.

POSTMORTEM DISAPPEARANCE OF GLYCOGEN IN THE NEW-BORN INFANT AND IN THE FOETUS. WILHELM HERTZ, Ztschr. f. Kinderh. **55**:410, 1933.

Hertz studied the postmortem disappearance of glycogen in the new-born infant and in the fetus.

LEVINSON, Chicago.

Acute Infections

THE BACTERIOLOGICAL DIAGNOSIS OF WHOOPING COUGH. NELLES SILVERTHORNE and DONALD T. FRASER, Canad. M. A. J. **32**:367 (April) 1935.

The results of the examination of three hundred and thirty-three children by the cough plate culture method are reported. The conclusions are: 1. The cough plate culture method is the most efficient means of establishing an early diagnosis of whooping cough. 2. Ninety-eight per cent of the cultures were positive in sixty-four children examined during the first week of the disease. 3. In two instances pure cultures were obtained on the original cough plates. Striking dissimilarities between freshly isolated and stock strains of the microorganism were found. One hundred and nineteen strains were isolated. Macroscopic agglutination tests were carried out. In every instance there were complete agglutination with the monovalent rabbit serum and no agglutination with the control serums.

The results of these studies suggest the desirability of selecting strains of Haemophilus pertussis for the preparation of vaccine which strictly conform to those qualities that characterize freshly isolated strains. MOORE, Omaha.

BRUCELLOSIS. O. B. MAYER, J. South Carolina M. A. **31**:99 (May) 1935.

Mayer reviews this disease from a historical standpoint, giving a brief account of Evans' work in proving that Micrococcus melitensis and Bacillus abortus are the same. She was recovering from a sickness herself when her blood agglutinated the brucella organism. Three types were recognized: bovine, porcine and caprine. Transmission of infection is by ingestion or through the skin or the eye. The symptoms uniformly present are malaise, aching and loss of appetite. The laboratory findings are of no import, except the agglutination reaction. No specific treatment is given, but a serum introduced by Foshay is suggested.

Prevention is accomplished by using pasteurized milk and by proper control of herds, especially by attention to contaminated forage.

WESTON JR., Columbia, S. C.

A SURVEY OF TONSILLECTOMY AND ADENOIDECTOMY IN SCARLET FEVER. GEORGE P. HUNT, New England J. Med. **212**:665 (April 11) 1935.

Two comparable groups of cases of scarlet fever are presented. Tonsillectomy and adenoidectomy were done on 273 patients, 215 of whom were operated on early in the disease and had no complications. Of the remaining 58 who were operated on, 27 entered the hospital with some complication, and 31 showed the complication while under hospital care, yet but twelve complications followed tonsillectomy. Ninety-nine of the nontonsillectomized group of 285 patients showed complications.

The earlier the operation is done, the less is the probability of complications and the shorter is the contagious period.

Fifty-six private patients are included in the early group of those with no complications, tonsillectomy having been done from the second to the sixth day of the illness. The majority of these private patients entered the hospital on the morning of the operation, returned home the same night and were kept in quarantine for the remainder of the required twenty-eight days. There has never been a second case in these families, nor does Hunt know of another contact case occurring in any family from any patient tonsillectomized in this series.

The mortality for the entire group of 558 patients consisted of three deaths, one from acute nephritis on the day of entrance and one following bilateral mastoidectomy on the fourteenth day of the illness, no tonsillectomy having been done. The third child died of bronchopneumonia on the fourteenth day, tonsillectomy and adenoidectomy having been done on the previous day.

GENGENBACH, Denver.

CURRENT PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES. Pub. Health Rep. **50**:633 (May 10) 1935.

For the period of March 24 to April 20, 1935, the number of cases of measles rose from 132,000 to 142,000. Compared with the incidence in recent years, this is about a 10 per cent increase. The number of cases of scarlet fever reached a peak of 8,495 at March 30 and has since rapidly declined. The current incidence of meningococcic meningitis is about three times that of 1934. Influenza, diphtheria and poliomyelitis show a decline. The average mortality for the period was 12 per thousand, which is normal. SANFORD, Chicago.

TREATMENT OF DIPHTHERIA. ALEXANDER JOE, Brit. M. J. **1**:711 (April 6) 1935.

The author presents a general discussion, with a consideration of the general management, the specific treatment and other subjects relating to diphtheria in all its phases.

ROYSTER, University, Va.

COLIBACILLUS INFECTIONS IN THE CHILD. G. MOURIQUAND and J. SCHOEN, J. de méd. de Paris **16**:358 (April 18) 1935.

The authors point out that the colon bacillus may be found in the urine without pathologic implication. Only its presence in large numbers in fresh aseptically procured urine is of significance. In the blood it is pathologic. Septicemia due to the colon bacillus may lead to localization in the meninges, respiratory tract and heart, but most frequently the kidneys. If the latter infection becomes chronic, it is important to search for congenital malformations.

LESLIE, Evanston, Ill.

PERSISTENCE OF DIPHTHEROTOXIN IN THE SUBCUTANEOUS TISSUE OF GUINEA-PIGS. D. D'ANTONA and M. VALENSIN, Pediatria **43**:431 (April) 1935.

Diphtheria toxin injected subcutaneously in guinea-pigs remains active for a long time, and its absorption differs in each subject. The toxin can be demonstrated in the edematous subcutaneous tissue by injection of the exudate into the dermal tissues of rabbits. The intradermal reactions thus provoked are identified as specifically diphtherotoxic by their characteristic appearance, by their inactivation by heat and by their disappearance in the presence of antitoxin.

SIGNORELLI, New Orleans.

PERMEABILITY OF THE MENINGES TO BROMIDE AND TO DIPHTHERIA ANTITOXIN IN THE COURSE OF DIPHTHERIA. V. MENGOLI, Riv. di clin. pediat. **33**:462 (April) 1935.

In cases of diphtheria, bromide will pass from the blood into the spinal fluid at a normal rate.

The spinal fluid of patients with diphtheria on the sixth day of the disease usually contains from $\frac{1}{100}$ to $\frac{1}{200}$ unit of diphtheria antitoxin per cubic centimeter; at the same time, the blood contains from 3 to 8 units per cubic centimeter. Appreciable amounts of diphtheria antitoxin are found in the spinal fluid twenty-four hours after an intravenous injection of the antitoxin.

Among seven cases of severe diphtheria, in only one was there found to be an increased protein content in the spinal fluid; in each case, however, the spinal fluid pressure was increased.

HIGGINS, Boston.

SERUM THERAPY FROM THE HEMATOLOGIC POINT OF VIEW: A STUDY OF THE RELATIONSHIP BETWEEN THE HEMATOLOGIC AND CLINICAL FINDINGS IN DIPHTHERIA. JEAN LANGE, Acta pædiat. **17**:238, 1934.

This is a long paper which fills sixty octavo pages. It presents in detail the results of 382 leukocyte counts and 319 differential tabulations on thirty-eight patients with diphtheria, also the data of 79 white blood cell counts and 49 differential counts on seven other patients suffering from nondiphtherial angina and on seven diphtheria carriers. The patients in all three groups were given diphtheria antitoxin. The blood of each patient was examined several times, viz., just before serum was administered, on the morning of the following day, on the morning of the third or fourth day thereafter, during the course of serum disease or at a time when serum disease was expected and, finally, once or twice during convalescence.

Although diphtheria tends to produce polymorphonuclear leukocytosis, there was no constant parallelism between the severity of the pharyngeal type of the disease and the total number of white blood cells or the "shift to the left" in the Schilling count. In the majority of cases absolute monocytosis existed, whereas the number of lymphocytes was generally unchanged; the number of eosinophilic leukocytes was also unaffected. In nasal diphtheria the total number of white cells was more dependable as an index of the severity of the infection.

In the case of diphtheria carriers the number of leukocytes was sometimes increased and sometimes remained stationary after the injection of serum. On the contrary, in twenty-one of thirty-eight patients with clinical diphtheria there was a critical fall in the white blood cell count after serum was administered. This observation led to the conclusion that failure of the white count to drop is an indication for the administration of more antitoxin. Specific antiserum produced a similar effect on the number of leukocytes in several patients with severe non-diphtherial pharyngitis; this illustrates the beneficial effect of nonspecific foreign protein. Diminution of the number of leukocytes was accompanied by decline in the concentration of neutrophilic cells; however, the shift to the left was not reversed until later in the period of recovery. Monocytosis was usually inconspicuous following the injection of serum, probably because the antitoxin conferred passive immunity, which obviated the need for active measures of defense of which monocytosis is thought to be an expression. The injection of serum was followed consistently by the appearance in the blood stream of plasma cells and of Türk cells. These were present in maximum concentration at the height of the serum disease. Serum sickness was characterized by leukocytosis involving both neutrophils and lymphocytes. Most of the patients exhibited eosinophilia two or three days after they had received serum; eosinophilia was most marked in those patients in whom serum disease did not develop.

One patient who died of malignant diphtheria soon after the injection of serum exhibited marked neutrophilic leukocytosis with a predominance of immature cells. The blood picture was not significantly abnormal in the case of five patients in whom cardiac complications developed, except that all showed a marked shift to the left before serum was given.

In order to insure safety in the employment of the huge doses of antitoxin which are indicated in cases of malignant diphtheria it is necessary to use a highly concentrated serum. Otherwise the ratio of units of antitoxin to the quantity of phenol present in the serum as a preservative is such that poisoning may ensue from an excessive dose of the bacteriostatic agent. McCune, New York.

Chronic Infections

Juvenile Paretic Neurosyphilis Studies: VII. The Descriptive Mental Picture. William C. Menninger, Am. J. Psychiat. **91**:1413 (May) 1935.

This is the seventh of a series of papers on juvenile dementia paralytica. In this discussion the author describes symptom complexes of the disease to illustrate the various predominating types. Jahr, Omaha.

Juvenile Paretic Neurosyphilis Studies: I. Incidence, Sex, and Age at Onset. William C. Menninger, Am. J. Syph. & Neurol. **18**:486 (Oct.) 1934.

On the basis of 43 cases of juvenile dementia paralytica in which personal observation was made and 610 cases collected from the literature, the following points are established regarding the incidence, distribution between the sexes and age at onset:

Cases of juvenile dementia paralytica represent less than 2 per cent of all cases of dementia paralytica. There are no reliable data to indicate either the frequency of occurrence of late congenital neurosyphilis in association with congenital syphilis (especially if the asymptomatic form is included) or that of juvenile dementia

paralytica in cases of late congenital neurosyphilis. Conservatively it can be estimated that in less than 10 per cent of the cases of congenital syphilis late neurosyphilis develops, that in from 5 to 15 per cent of the cases of late congenital neurosyphilis the disease is manifested as juvenile dementia paralytica and that probably in less than 1 per cent of all cases of congenital syphilis dementia paralytica develops.

With inadequate data from which to judge it would appear that the percentage of cases of congenital syphilis in which dementia paralytica develops is smaller than that of cases of acquired syphilis in which dementia paralytica develops.

On the basis of 619 cases of juvenile dementia paralytica the ratio of males to females is 1.36:1, as compared with the ratio of 3.43:1 in cases of the acquired (adult) form of the disease.

The average age at onset in 273 males was 13 years, and in 213 females, 12.9 years. The time of onset, as stated in 542 cases, was between the ninth and the eighteenth year in 68 per cent of the cases.

Onset before the sixth year occurred in 9.4 per cent of the cases. This group includes cases in which the patients were mentally and physically inferior from birth and showed no gross change at any age which might be regarded as the onset of the disease. More correctly, the children might be regarded as having infantile dementia paralytica.

The onset occurred after the twentieth year in 3.6 per cent of the cases, including 5 cases in which it occurred after the twenty-fifth year. As far as one is able to judge, these are authentic cases of the juvenile dementia paralytica form of neurosyphilis in that the dementia paralytica appeared as a late manifestation of congenital syphilis.

The average age at examination of 278 males was 16.53 years and of 197 females, 16.11 years. There is, then, an average interval of 3.53 years for males and of 3.21 years for females between the time of onset and that of examination.

AUTHOR'S SUMMARY.

JUVENILE PARETIC NEUROSYPHILIS STUDIES. WILLIAM C. MENNINGER, J. Lab. & Clin. Med. **20**:806 (May) 1935.

Menninger gives the following review of the laboratory findings in a group of three hundred and twenty-five cases of congenital neurosyphilis. The Wassermann reaction of the blood was negative in seven cases, of the spinal fluid in six cases and of both the spinal fluid and blood in two cases. The cell counts and colloidal gold curves were essentially similar to those in cases of acquired neurosyphilis.

The author concludes that a diagnosis of congenital neurosyphilis is justified when the history, stigmas, clinical signs and symptoms all point to it, even though all the laboratory findings are negative. McMURRAY, Galveston, Texas.

JUVENILE PARETIC NEUROSYPHILIS STUDIES: X. CLINICAL COURSE, INCLUDING PRODROMAL SYMPTOMS, NATURE OF ONSET, REMISSIONS, AND DURATION. WILLIAM C. MENNINGER, Am. J. Syph. & Neurol. **19**:238 (April) 1935.

A continuation of the studies by this author is reported. The statistical element makes the article difficult to abstract. ROYSTER, University, Va.

JUVENILE PARETIC NEUROSYPHILIS STUDIES: XI. TREATMENT. WILLIAM C. MENNINGER, Am. J. Syph. & Neurol. **19**:257 (April) 1935.

A continuation of the studies by this author is reported.

ROYSTER, University, Va.

INTRACUTANEOUS (MANTOUX) TUBERCULIN REACTION: A COMPARATIVE STUDY OF POSITIVE AND NEGATIVE REACTORS. MEYER PARETZKY, Am. Rev. Tuberc. **31**:553 (May) 1935.

This is a study of two almost equal groups of patients, the one giving positive and the other negative reactions to tuberculin, who belonged to families in which the

hygienic milieu and ways of living were comparable. Careful subsequent observations were made for periods of months and years to determine the developments in the two groups. Analysis of the findings on subsequent examination showed that both the diagnosis and the prognosis were considerably more unfavorable for the negative group. The negative reactors that remained nontuberculous were either removed from the source of infection or had acquired a positive reaction to tuberculin. The "dosage" of the infection and the length of exposure were found to be the principal factors which had influenced the development of disease in the negative reactors who later showed evidence of tuberculous disease.

EUGENE SMITH, Ogden, Utah.

THE TUBERCULOSIS OF CHILDHOOD. CHARLES HENDEE SMITH, Ann. Int. Med. **8**:1090 (March) 1935.

In this concise article many of the prevalent misconceptions of tuberculosis in children are corrected. The pathogenesis of the disease is convincingly discussed and correlated with the clinical types. The course which the disease may take after the primary infection is clearly explained.

The relative importance of the diagnostic procedures is stressed. In the diagnosis of tuberculosis three questions must be kept in mind: (1) Has the child been infected with tuberculosis? (2) What is the site of the disease? (3) Is the disease active, stationary or cured?

The clinical types of tuberculosis occurring at different age periods, the incidence and the treatment of the disease in children are discussed.

READING, Galveston, Texas.

FATALITY FROM EXACERBATION OF LATENT TUBERCULOSIS DUE TO THIOBISMOL IN A CASE OF YAWS. C. M. HASSELMANN, Arch. Dermat. & Syph. **31**:686 (May) 1935.

A girl of 9 years was apparently healthy except for the generalized polypapilloma of yaws. She was treated with injections of one dose each of bismuth hydroxide dispersed in oil and a suspension of bismuth salicylate in oil, with an interval of three days. Three days later she was given 0.2 Gm. of thio-bismol. Two days later fever developed and there was scanty urination. Death followed in four days. Autopsy showed tuberculosis of the lungs and cervical lymph nodes, fatty degeneration of the liver and tubular nephrosis. The author reemphasizes the superiority of arsenic in the treatment of yaws and believes that if bismuth must be used in certain cases the oily suspensions with the so-called deposit action are preferable to water-soluble preparations and that the former are less toxic and much less likely to produce acute exacerbations of latent tuberculosis.

DULIN, Iowa City.

THE CONTROL OF TUBERCULOSIS IN CHILDHOOD. J. W. AMESSE, South. M. J. **28**:276 (March) 1935.

The present day measures for the control and eradication of childhood tuberculosis are briefly enumerated as follows:

1. Development of organized agencies, national, state and local.

2. Segregation of patients with active tuberculosis in sanatoriums or hospitals.

3. Protection of the food supply, including the testing of cattle, pasteurization of milk, inspection of meat, examination of food handlers and screening of foods exposed for sale.

4. Examination of school children, including not only the physical survey but the use of intradermal tests and roentgenography and, particularly, a follow-up in the homes of the children found to be infected.

5. In close association with this great movement, the evolution of the open air or sun school, the summer camp and the preventorium and the general employment, in the physical training of classes in grade-school, of breathing exercises directed toward more complete expansion of the lungs.

6. As a natural corollary of such protective measures among the young, the educational campaign carried out by officials and social groups for the information of parents and guardians. While this vital activity is advancing in ever increasing volume, it frequently lacks the direct appeal necessary to produce cooperation.

SCHLUTZ, Chicago.

WHY THE PULMONARY FORM OF TUBERCULOSIS IS COMMON IN ADULTS AND THE NON-PULMONARY FORM IN CHILDREN. P. F. ARMAND-DELILLE, Brit. J. Tuberc. **29**:64 (April) 1935.

According to the statistics of Armand-Delille two thirds of the infants and children in contact with tuberculous parents die of the acute forms of the disease, i.e., miliary tuberculosis or tuberculous meningitis, and in only one third do chronic localizations develop. Many of the children in the latter group appear clinically sound but show a positive Pirquet reaction. In this group, as evidenced by the cutaneous reaction, some degree of immunity has been established, and the infection tends to remain localized. In the children who die, the dose and virulence of the infecting organisms overwhelm the subject before immunity is established. Certain children acquire only a partial immunity as a result of the first infection, and when reinfection occurs localized tuberculosis develops, such as Pott's disease and tuberculosis of the cervical glands, kidneys, liver, etc. In children the organs which are developing most rapidly appear specially to be loci minoris resistentiae to the bacillus. In the child with a primary infection in the lung, the focus may become calcified and remain dormant for many years. A special period of danger arises, however, during adolescence. At this time, especially in girls, rapid decalcification occurs, and this, aided by lowered general resistance due to overwork or study, may permit reactivation of the old infection and the development of pulmonary tuberculosis. It appears probable that localization in the lung is seldom due to recent contact with carriers of bacilli but that it follows reactivation of an old infection contracted many years before.

Once the period of childhood is past, the older an adult the more resistant he becomes to tuberculosis. In the adult also the lung seems to be the organ that offers the least resistance to tuberculosis, perhaps because the capillary system constitutes a screen for all the tubercle bacilli carried by the blood stream. By this time also, with the exception of the kidneys and genital organs, most of the tissues of the body have acquired a special resistance to the infection.

EUGENE SMITH, Ogden, Utah.

ACTINOMYCOSIS OF LUNG. T. S. RODGERS, Proc. Roy. Soc. Med. **28**:831 (May) 1935.

The case was reported first in 1933, when intensive iodide therapy was started. The child took as much as 120 grains (7.78 Gm.) a day. The respiratory rate and temperature fell to normal levels in a few days. The wound in the chest continued to drain small quantities of serosanguineous discharge for from six to eight weeks and then healed. The general health improved slowly during the past two years. Except for slight dyspnea on exertion the boy is free from symptoms. The physical signs in the chest remained unchanged for the first year. Since that time there has been considerable improvement in both the roentgenographic and the physical signs. The clubbing of the fingers was definitely less marked.

The last roentgenogram, taken in February 1935, showed marked clearing of the right lower lobe with residual fibrosis at the base, pleural thickening and slight displacement of the heart and trachea to the right.

WILLIAMSON, New Orleans.

CASE OF CONGENITAL TUBERCULOSIS. H. GRÉNET, M. METZGER, A. HÉRAUX and J. MÉZARD, Arch. de méd. d. enf. **38**:229 (April) 1935.

The history in the case of transplacental infection reported includes that of the mother, aged 23, who entered the hospital in the eighth month of pregnancy, suffering from severe cachexia. The infant, although small (2,600 Gm.) seemed normal. On account of the maternal infection, the baby was given BCG orally on the second, fifth and seventh day of life, and in addition mercurial friction rubs were employed, together with ultraviolet irradiation. In spite of careful nursing, the infant gained weight very slowly; in the second month diarrhea developed, with irregular fever; vomiting followed; there was rapid loss of weight, and disseminated râles were noted in both lungs. Both the liver and the spleen became involved, but the more common signs of congenital syphilis were absent. The cutireaction to tuberculin remained negative. The infant died on the sixty-fifth day.

Autopsy disclosed generalized tuberculosis with an extraordinary abundance of Koch's bacilli. Inoculations were made at the Pasteur Institute, with positive results.

AMESSE, Denver.

DEFORMITY OF THE TIBIAS IN A CASE OF HEREDITARY SYPHILIS. L. BABONNEIX, MACÉ and MME. JOURDAN, Bull. Soc. de pédiat. de Paris **32**:521 (Nov.) 1934.

A boy, 10 years old, showed marked progressive deformities of the tibias. The right tibia presented the appearance of a saber shin, the thickening being present in the middle third of the anterior part of the bone while the left tibia had two areas of thickening along the anterior surface, one just below the tuberosity and the other of the lower third, the middle third of the bone being uninvolved. Roentgenograms confirmed the presence of marked changes in the periosteum as well as areas of condensation and rarefaction in the medullary parts. The swellings were somewhat tender, and the skin over the most prominent part of the right tibia was eroded. The boy had other signs suggesting hereditary syphilis, and the Wassermann reaction of the blood was strongly positive.

BENJAMIN, Montreal, Canada.

PRIMARY TUBERCULOUS CUTANEOUS INOCULATION AND REGIONAL ADENITIS TREATED SURGICALLY. M. COFFIN, Bull. Soc. de pédiat. de Paris **32**:535 (Nov.) 1934.

In a girl, 7 years old, inguinal adenitis developed after a wound on the knee, which became infected and left a violet paper-like scar. A number of inguinal glands were originally involved, but the condition subsided in all except one, in which it progressed until the tissue broke down. The glands were excised, as well as the area of unhealthy skin. Histologically tubercles were observed in the glands, and bacilli were demonstrated in them. The skin showed typical epithelioid and giant cells with involvement of a sweat gland. The child had been in contact with the father, who had tuberculosis. The reaction to the tuberculin test was positive. It is considered that in this case there was a primary tuberculous lesion of the skin, the bacilli having entered by way of a wound, and that the inguinal glands became involved by extension up the leg of bacilli through the lymphatic channels. Primary tuberculosis of the skin is serious in infants, who may succumb to the infection, but in older children the prognosis is much better. When the skin is secondarily involved by tubercle bacilli which originate in other foci in the body, the lymph glands in the region of the infected skin do not usually caseate.

BENJAMIN, Montreal, Canada.

RÔLE OF MINERAL METABOLISM IN INCIPIENT TUBERCULOSIS OF CHILDREN. G. POPOVICIU, Compt. rend. Soc. de biol. **116**:1101 (April 20) 1934.

The calcium and phosphorus factors have been shown to be definitely increased in the blood in cases of early or incipient tuberculosis, whereas they are lowered to normal levels during healing or in cases of markedly grave disease.

The author studied the calcium and phosphorus contents of the blood of tuberculous small children and infants in both the summer and the winter. It was found that the calcium and phosphorus content in the normal children was on the average 45.1 mg. per hundred cubic centimeters in summer and 36.1 mg. in winter, as compared with 46.4 and 49.6 mg., respectively, in the tuberculous children. The increase in the content for normal children during the summer undoubtedly is due to the effect of the rays of the sun.

In addition, Popoviciu found the calcium-phosphorus product (calcium content multiplied by the phosphorus content) to be lowered below the normal figure in cases of progressive tuberculosis, such as tuberculous osteomyelitis. With treatment the values again rose when healing occurred, as shown by 34.7 mg. per hundred cubic centimeters before and 55.8 mg. after treatment.

The increase in the calcium-phosphorus product is the result of a defense mechanism, i.e., the mobilization of lime salts to aid in healing. This progress can be interpreted either as one of demineralization and devitamination or as a reestablishment of the normal equilibrium.

WAHL, Cleveland.

CONGENITAL PULMONARY STENOSIS AND PULMONARY TUBERCULOSIS. C. AUBERTIN, Paris méd. **1**:413 (May 4) 1935.

A case of pulmonary tuberculosis in a boy, aged 15, suffering from congenital pulmonary stenosis is reported. The boy's mother had died of tuberculosis and was also said to have had a cardiac lesion. The literature is discussed.

LESLIE, Evanston, Ill.

ERYTHEMA NODOSUM ASSOCIATED WITH A NEGATIVE REACTION TO TUBERCULIN. NILS LANDORF, Rev. franç. de pédiat. **11**:157, 1935.

Seventeen cases of erythema nodosum are cited in which, in spite of large doses, the reaction to tuberculin remained negative. A case of special interest occurred in a girl, aged 7 years, who had two episodes of erythema nodosum six months apart. At the time of the first episode, the tuberculin reaction was negative with as high as 3 mg. of tuberculin, nor was there any clinical or roentgenologic evidence of tuberculosis. With the second episode the tuberculin reaction was markedly positive with 0.1 mg., and the roentgenogram showed a primary tuberculous lesion in the upper lobe of the left lung. From these observations one must conclude that erythema nodosum may be a nonspecific allergic symptom in spite of its usual association with tuberculosis.

LESLIE, Evanston, Ill.

TUBERCULOUS LARYNGITIS IN A CHILD. B. PINCHERLE, Pediatria **43**:576 (May) 1935.

A case of tuberculous laryngitis in a boy of 3½ years is reported, with a review of the literature. The patient exhibited extensive pulmonary exudative lesions and a tuberculous lesion of the larynx of the ulcero-infiltrative type. Though the Pirquet and Mantoux reactions were negative, the tubercle bacillus was recovered from the mucosa of the larynx.

SIGNORELLI, New Orleans.

GERSON-HERRMANNSDORFER-SAUERBRUCH DIET IN THE TREATMENT OF CHILDHOOD TUBERULOSIS. A. GOLIN and R. DEMENIGHINI, Riv. di clin. pediat. **33**:257 (March) 1935.

Thirty children between the ages of 3 and 9 years, having various types of tuberculosis, were given a diet patterned after those of Gerson and Herrmannsdorfer and Sauerbruch. The diet is definitely low in sodium chloride and high in vegetables and fruits. A sample diet for a 6 year old child weighing 43 pounds (19.5 Kg.) contained the following foods:

Milk, 500 Gm.; meat, 80 Gm.; vegetable purées, 100 Gm.; raw or steamed greens, 100 Gm.; cooked fruit, 100 Gm.; raw fruit, 100 Gm.; macaroni, 20 Gm.; bread, salt-free, 40 Gm.; butter, 20 Gm.; cod liver oil, 40 Gm., and olive oil, 20 Gm.

The values of the diet were as follows: protein, 2.6 Gm.; fat, 6 Gm., and carbohydrate, 7.4 Gm., per kilogram of body weight.

Sixteen of the thirty children did excellently, and nine others, fairly well, when they were fed the diet; it led not only to improved nutrition but to clinical improvement. The other five children did not do well clinically, although several showed a gain in weight and an increase in the hemoglobin concentration.

HIGGINS, Boston.

MONOCYTES IN TUBERCULOSIS OF CHILDREN. G. B. SEGHEZZO, Riv. di clin. pediat. **33**:313 (March) 1935.

The author studied the blood counts of sixty-eight children with various types of tuberculosis. In mild tuberculosis an increase in the monocyte count does not occur regularly. In severe forms the monocyte count is increased, while the ratio of lymphocytes to monocytes is decreased.

Monocytosis is of little or no value in the diagnosis of tuberculosis of children but is of proved value in prognosis, monocytosis indicating an unfavorable outcome.

HIGGINS, Boston.

PROPHYLAXIS OF TUBERCULOSIS IN INFANCY. GONZÁLEZ ALVAREZ, An. de pediat. **17**:299 (May) 1935.

To prevent infection and increase the power of resistance of the child to the disease is the dual aim in the prophylaxis of infantile tuberculosis. There should be isolation of the sick or of the well and, at all events, separation of the child from any focus of infection. Exposure to infection occurs in groups of children, such as those in schools, asylums, nurseries, etc., and from teachers and nursery-maids with the disease. In one class to which a professor with open tuberculosis gave lectures during one year, all the students became infected; in another school (Braenning) 40 per cent of the children gave positive reactions to tuberculin on entering at the age of 6 years and 80 per cent at the age of 15 years. The tuberculin test should be made on every infant of 6 months or over. The finding of a positive reaction leads to strict investigation of all persons who surround the child and to the discovery, not infrequently, of a spreader of bacilli till then unrecognized.

Removal of infants and young children from tuberculous surroundings to homes in the country, under medical and nursing supervision, is advocated. Recently, of 18,000 children of tuberculous parents thus placed and aided by the Grancher Agency, tuberculosis developed in only 15. When it is impractical to remove the child from contagion, immunization by use of the Calmette-Guérin (BCG) vaccine is strongly advocated. This vaccine is supplied free on prescription by certain Spanish laboratories, which make it from the original bacilli presented by Dr. Calmette himself.

GIFFEN, Toledo, Ohio.

DIAGNOSIS OF OSTEO-ARTICULAR TUBERCULOSIS IN THE INFANT. URBANO GONZÁLEZ, Rev. chilena de pediat. **6**:159 (April) 1935.

In a case in which tuberculous osteo-arthritis is suspected the author advises aspiration of the lesion in the hope that pus or liquid material may be obtained for examination for Koch bacilli. If this fails a biopsy of tissue from the suspected lesion may give the required information. Both these procedures may fail. The author advocates a method as a substitute, which consists in a biopsy of tissue from lymphatic glands situated near the suspected lesion. Commonly the Koch bacillus will be seen in such a gland. The marked predilection of this organism for lymphoid tissue suggested this method to the author. In his opinion, lymphatic glands adjacent to any tuberculous lesion will always be found to be involved, and they will almost certainly contain active tubercle bacilli.

SCHULTZ, Chicago.

ERYTHEMA NODOSUM AND TUBERCULOSIS. H. MÜLLER, München. med. Wchnschr. **82**:820 (May 23) 1935.

Erythema nodosum occurs more frequently in children than in adults. Müller reports two cases in adults aged 20 and 21. Both patients had active pulmonary tuberculosis, and one also had extrapulmonary lesions. After a year one of the patients had a recurrence of the erythema nodosum with a simultaneous reactivation of the pulmonary process.

BRAHDY, Mount Vernon, N. Y.

DIAGNOSIS OF HEREDITARY SYPHILIS IN NEUROPSYCHIATRIC DISTURBANCES OF CHILDHOOD. ERICH STERN, Ztschr. f. Kinderpsychiat. **2**:2 (April) 1935.

Among the causes of disturbances of the mind two factors are of outstanding importance—alcoholism and syphilis. Both these factors not only operate in the immediate family but extend to future generations (mongolism, epilepsy, Little's disease, psychopathic personality, etc.).

The importance of the early recognition of hereditary syphilis is obvious. Unfortunately, the physician often spends too much time looking for signs of degeneration and too little searching for stigmas which afford valuable clues if not pathognomonic signs of the disease early in childhood. A careful family history, especially with reference to prenatal syphilis, death from circulatory disturbances (aortic aneurysm), the early death of a parent, apoplexy, paralysis, tabes dorsalis, etc., is worthy of scrutiny. As to the patient himself, the neonatal history and the criteria of early development with reference to physical and mental progress, with particular stress on motor control, are of great importance in establishing corroborative data pointing to evidence of syphilitic infection. A history of convulsions, whether attributable to injury at birth, illness or "teething," must be carefully evaluated. Sphincter control or its absence must be studied.

Examination of the teeth and gums may at times offer evidence of syphilitic infection. Caries of the temporary (or milk) set, erosion of the gums and deformities of various types must not be taken lightly. Hutchinson teeth should not be unrecognized.

Deformities of the skull (craniotabes), unless due specifically to rickets, are of great significance. Roentgenographic studies of other bones (the sternum and sacrum) at times reveal evidence of syphilis, which with other criteria may establish the diagnosis early. If to these data one adds difficulty in hearing without definitely accountable causes, rhagades about the mouth, saddle nose, mongolian-like epicanthus, cardiac anomalies and anomalies of the sex organs and the entire endocrine system, a timely diagnosis of hereditary syphilis becomes a matter of careful study. A negative Wassermann reaction is not conclusive in ruling out congenital syphilis.

The author concludes that even if all the data point only to a probable diagnosis of syphilis in the child, the seriousness of the situation (to the individual and the community) is such that institution of vigorous antisyphilitic treatment is justified.

JAHR, Omaha.

CASE OF TUBERCULOUS MENINGITIS IN AN INFANT WITH THE NONNE-FROIN SYNDROME. MARIA GUTMANOWA, Pedjatrja polska **14**:205, 1934.

Gutmanowa describes a case of tuberculous meningitis in a 6 month old baby in which spinal puncture yielded xanthochromic fluid but in which the fluid in the ventricles was clear and under pressure. This condition is called the Nonne-Froin syndrome and is due to prolonged cisternal block.

KRUPINSKI, Chicago.

OBSERVATIONS ON THE COURSE OF TUBERCULOUS INFECTION IN A PAIR OF DIFFERENTLY NURTURED TWINS. I. JUNDELL, Acta pædiat. **17**:131, 1934.

A pair of binovular twins contracted a tuberculous infection at the age of 9½ months. Two months later each was fed a diet differing from that of the other.

Twin A received a mixed diet with cream in addition; twin B was given daily from 500 to 600 cc. of soured skimmed milk, carbohydrate and raw fruit. At the beginning of the experiment the physical condition of twin A was better than that of his brother. At the end of eight months this relation was reversed. At this time twin B was also given a mixed diet with added cream. Nevertheless, he continued to display physical superiority over his brother. Repeated roentgenographic examination revealed no difference between the two infants as regards the evolution of the tuberculous infection. Jundell draws the cautious conclusion that "the sour milk-fruit diet was not inferior to the general diet."

McCune, New York.

Internal Diseases

Dental Extraction in Hemophilia. Walter William Dalitsch, J. Am. Dent. A. **21**:1804 (Oct.) 1934.

The article is based on observations made on a series of the hemophiliac patients of Dr. Carrol Birch of the University of Illinois. "Bleeding from the oral tissues, either spontaneously or as a result of dental procedures, was found to be a frequent occurrence." The most serious bleeding and the most difficult to control was that following tooth extraction.

Management in these cases included: avoiding extractions unless the clotting time was twenty minutes or less; administration of rather large doses of ovarian extract; selection of a time in the periodic bleeding cycle when the coagulation time was lowest; of a close fitting molded vulcanite splint to protect the clot and limit the escape of tissue secretions; application of ovarian extract at the site of bleeding.

Three cases are reported.

Noyes, Chicago.

Portal Cirrhosis in a Child. Wilfred Sheldon, Proc. Soc. Roy. Med. **28**:761 (April) 1935.

A case of this condition in a boy 4½ years of age is reported.

Williamson, New Orleans

Some Observations on the Aetiology and Effect of Alkalis on the Nephrotic Syndrome. R. B. Hawes and E. C. Vardy, Quart. J. Med. **4**:1 (Jan.) 1935.

Hawes and Vardy studied a group of patients admitted to the Tan Tock Seng Clinic in Singapore from November 1929 to April 1933. This group consisted of forty-three patients showing the essential findings of the nephrotic or of the nephrosonephritic type of syndrome.

Alternating acid-forming and base-forming diets were used, ammonium chloride being given with the former and an alkaline mixture of potassium bicarbonate and potassium citrate being administered with the latter.

Ankylostomiasis and malaria were not found to be contributing to the causation of nephrosis in this group of Chinese. Diet seemed to be important. The authors found that an alkaline diet produced improvement; an acid diet increased the symptoms. Potassium salts, in addition to the special diet, completely controlled the edema, the hypercholesteremia and, to a certain extent, the albuminuria.

In addition to the dietary factor, infection was thought to play a part. Some of the edematous patients reacted clinically to hemolytic streptococcic nucleoprotein, which led the authors to suspect Streptococcus haemolyticus as a possible infecting agent.

Bradford, Rochester, N. Y.

A Rare Form of Familial Osseous Dystrophy. L. Morquio, Arch. de méd. d. enf. **38**:5 (Jan.) 1935.

Since Morquio described the unusual type of dystrophy which has now become associated with his name, sixteen further cases have been reported in the literature.

In his original communication the author discussed the clinical features of two members in a family of five, and in his present contribution he relates in great detail the history of two other members of this family, the fifth child being entirely normal.

All four of these brothers presented identical deformities: There were gross alterations in growth and in structure and accentuated osteoporosis with the same onset and the same evolution; all these deformities were completely symmetrical. The thorax was shortened and broadened, with the sternum protuberant, resembling that in Pott's disease. There were no deformities of the skull, however, and the patients had normal intelligence. The disease attacks primarily the motor system, is familial, begins in early infancy with a hereditary or constitutional pathogenesis and must be differentiated from chondrodystrophy.

AMESSE, Denver.

APPENDICITIS AND THE ACUTE ABDOMINAL SYNDROME DUE TO PURPURA. JACQUES LEVEUF, Bull. Soc. de pédiat. de Paris **31**:466 (Nov.) 1933.

A girl, 6½ years old, was admitted to the hospital suffering from acute abdominal pain and purpura. Examination of the blood revealed the findings of purpura haemorrhagica. The abdomen showed a slight rigidity of the right lower quadrant, where a purpuric patch was present. There had been leukopenia on a previous examination, when the child had no abdominal pain, but now there was polymorphonuclear leukocytosis. Operation was postponed at first because of the presence of purpura, but when the abdomen was opened a few days later a perforated gangrenous appendix and peritonitis were found. The child did well for a short time, but soon severe hemorrhage into the wound as well as from the nose developed. A splenectomy was then done, but the child died immediately afterward. The liver and spleen were not palpable in this case. A sister of this child had died of hemorrhage due to purpura haemorrhagica. The differential diagnosis of appendicitis in a child with purpura is discussed, and evidence is cited that in cases in which the appendix has been removed, even though it was found to be not inflamed, the child usually survived. It is emphasized that the spleen should be removed in cases of true purpura haemorrhagica before severe anemia as a result of hemorrhage develops, as when the operation is performed late the results are poor.

BENJAMIN, Montreal, Canada.

TREATMENT OF BRONCHOPNEUMONIA IN INFANTS UNDER ONE YEAR OLD. B. WEILL-HALLÉ and RUIN, Bull. Soc. de pédiat. de Paris **31**:473 (Nov.) 1933.

The infants treated were divided into four groups. The percentage mortality was highest for the infants from birth to 3 months old; there was a decrease in the mortality rate for the groups from 3 to 6 and 6 to 9 months old; for the group from 9 to 12 months old the mortality rate rose, as compared with the previous two groups. In each group some of the infants received only the routine treatment, which included mustard plasters and cupping; a second series were given intramuscular injections of a colloidal gold preparation every second day until the fever subsided, and a third series were given the routine treatment, together with injections of colloidal gold as well as injections of eucalyptene on alternate days. The latter were frequently followed by the formation of local abscesses. The mortality rate was highest for the first series and lowest for the third group. The number of cases reviewed is not large and covers three winter periods.

BENJAMIN, Montreal, Canada.

ABDOMINAL SYMPTOMS AT THE ONSET OF BOUILLAUD'S DISEASE IN CHILDREN. P. GAUTIER, J. de méd. de Paris **55**:71 (Jan. 24) 1935.

Gautier reports a case of acute rheumatic fever with cardiac involvement; on the first three days there were sore throat and severe abdominal pain and tenderness, with distention and generalized rigidity. There were also some stiffness of the neck and a positive Kernig's sign. The appendix and peritoneum

appeared normal at operation. There was some pain in the right knee on the third day. The cardiac symptoms developed on the tenth and eleventh days. There was a systolic murmur at the apex with a pericardial friction rub. Treatment with salicylates was instituted, with gradual improvement. The mitral lesion persisted. Gautier cites six other cases from the literature in which acute rheumatic fever gave a similar picture at the onset. In two of these a tentative diagnosis of pneumococcic peritonitis was made.

LESLIE, Evanston, Ill.

PNEUMOCOCCIC NEPHRITIS. DE LÉOBARDY and LABESSE, J. de méd. de Paris **55**: 119 (Feb. 7) 1935.

The authors state that they have observed many cases of pneumococcic nephritis since 1919. They do not give exact figures, but express the belief that the condition is more frequent than is generally supposed. It is seen complicating other localized conditions due to the pneumococcus or in an apparently primary form. It is characterized by a period of oliguria lasting several days. The urine contains albumin and red cells. No mention is made of edema. The prognosis is good in children. In adults with the secondary form there was a 50 per cent mortality. Three cases of the primary form were reported, in all of which there were positive blood cultures; all the patients recovered.

LESLIE, Evanston, Ill.

AN EPIDEMIC OF CATARRHAL JAUNDICE. M. LOPEZ PONDAL, Nourrisson **23**: 17 (Jan.) 1935.

This report from Argentina covers seventeen cases in infants aged from 18 months to 5 years. The majority occurred in the group aged from 2 to 3 years. In most of the cases the disease ran a benign course. The etiologic possibilities are discussed, but the only positive finding was bacilluria due to the colon bacillus. Spirochetes were searched for but never found. The author favors the designation of infectious icterus as being more accurately descriptive than catarrhal jaundice.

MCCLELLAND, Cleveland.

PERICORONITIS OF THE FIRST MOLARS AS A CAUSE OF FEVER IN INFANTS. G. L. HALLEZ, Nourrisson **23**:30 (Jan.) 1935.

Another case is added to those already reported by Hallez of a baby with high fever the only cause that could be found for which was a collection of seropurulent secretion around the crown of one of the first premolar teeth. The baby was 12½ months old. Careful physical examination ruled out other sources for the fever and illness. After incision and evacuation of the seropurulent material the symptoms were relieved and the temperature promptly dropped to normal. The eruption of only the premolar teeth have been found to produce this type of pericoronitis and its attendant symptoms.

MCCLELLAND, Cleveland.

SLIGHT SIGNS OF THYROID INSUFFICIENCY. L. ABRICOSSOF, Rev. méd.-sociale de l'enf. **1**:17 (Jan.-Feb.) 1935.

The author mentions several slight signs of thyroid insufficiency which may be overlooked when appearing singly or not in a typical syndrome of hypothyroidism: pale, thick skin; localized edema of the eyelids or face; clammy hands, knotty fingers; dry, scanty or brittle hair; irregular teeth, persistent constipation, and certain nervous disorders such as sluggishness of mind, apathy, indifference, obstinate headaches, fatigue and a tendency to drowsiness. A lowered basal metabolic rate, of course, confirms the diagnosis. Single signs which may first attract the parents' attention are: a retarded height and weight, obesity or mental sluggishness. Thyroid extract given by mouth (together with other grandular products in cases of pluriglandular disturbance) will quickly alleviate the signs of hypothyroidism.

SHAW, Evanston, Ill.

OSTEOCHONDRITIS OF THE SHOULDER. I. MARIAN, Rev. d'orthop. **22**:36 (Jan.-Feb.) 1935.

Osteochondritis of the upper end of the humerus is not common. The author has collected only six instances of this condition from the European literature. The pathologic changes of the condition are still in doubt. In Marian's opinion, the course of events is an infection, followed by inflammation, congestion, resorption and, finally, reconstruction of bone.

RYAN, Vancouver, Canada.

A CASE OF HYPERTROPHIC PYLORIC STENOSIS. FERDINANDO BONIVENTO, Pediatria **43**:72 (Jan.) 1935.

A case of hypertrophic stenosis of the pylorus in a 2 month old child is reported. Bonivento emphasizes the importance of early diagnosis as an assurance of success in avoiding inanition which follows protracted medical treatment in these cases. The patient recovered following surgical intervention (Weber-Rammstedt operation).

SIGNORELLI, New Orleans.

ROENTGENOTHERAPY IN NEPHRITIS. G. BETTINARDI, Pediatria **43**:121 (Feb.) 1935.

Thirteen cases of acute, subacute and chronic nephritis in children treated with roentgen irradiation of the dorsolumbar region are reported. The author presents a review of the literature on the subject and adds his observations on the influence of the roentgen rays on renal tissue based on experimental work with rats. In the second part of the paper he discusses the technic and indications for renal roentgenotherapy, together with an explanation of the mechanism of the action of the roentgen rays in nephritis.

The ages of his patients ranged from 4 to 14 years, and the cases included (*a*) eight of acute glomerular nephritis, (*b*) four of subacute glomerular nephritis and (*c*) one of chronic nephritis (nephrosclerosis) with uremia.

Patients with acute glomerular nephritis showed the most satisfactory results—a uniform response, with a diminution of hematuria and albuminuria, increase in renal function and improvement of the general condition of the patient. In patients with subacute involvement there were only partial effects on hematuria, albuminuria and edema. In the only case of chronic nephritis with uremia no definite results were obtained.

The author states that irradiation of the dorsolumbar region produces no immediate or remote damage to the renal tissues in rat or in man and that the procedure has a certain beneficial action in some clinical forms of nephritis.

SIGNORELLI, New Orleans.

UNUSUAL ASPECTS OF BRONCHOPNEUMONIA OF CHILDREN. V. ZAMORANI, Riv. di clin. pediat. **33**:40 (Jan.) 1935.

Zamorani reports the cases of two infants with bronchopneumonia of an unusual type not described in the literature. The cases were characterized by the young age (3 months) of the infants, the lack of clinical signs pointing to involvement of the respiratory tract, the absence of fever, a long and benign course (forty days), a poor appetite and failure to gain in weight. Roentgen and physical examination placed the involvement in the hilar region; there were dulness and diminished breathing in the back between the scapula and the spines. In one of the cases a pneumococcus was obtained from the sputum; its type could not be determined. An autogenous vaccine was made and used in treatment.

HIGGINS, Boston.

A CASE OF NEPHROSIS IN A GIRL OF 20 MONTHS. E. INGLESSI, Riv. di clin. pediat. **33**:138 (Feb.) 1935.

Inglessi describes a case of lipoid nephrosis in a girl of 20 months. The etiology was not apparent; the child died of bronchopneumonia after one month's illness.

HIGGINS, Boston.

POLYHORMONAL SYNDROME IN A GIRL. N. GÁLVEZ FERMÍN, VICTOR FAJARDO CARPIO and JUAN JIMÉNEZ, Arch. de med. inf. **3**:813 (Dec.) 1934.

A girl 14 years of age suffered from alternate periods of metrorrhagia and amenorrhea. Such cases fall into the class of the polyhormonal syndromes described by Zondek. They call for a thorough gynecologic examination of the condition of the uterus and adenexa. Theelin will help establish the diagnosis. The authors believe that chronic alcoholism in the father of this child caused an organic stigma that fomented the impairment of the ovary. Pathologic examination showed an absence of corpus luteum. The treatment consists in eliminating the overproduction of theelin, which can be done by ovariectomy and enucleation of the follicular cyst with manual destruction, as recommended by Zondek.

SANFORD, Chicago.

TREATMENT OF PYURIA IN INFANCY. LAGES NETTO, J. de pediat., Rio de Janeiro **2**:72 (Feb.) 1935.

For this condition the author uses complete rest in bed, abundant fluids, milk as the principal item of diet, phenyl salicylate as a medicament and vaccines occasionally.

SANFORD, Chicago.

ABDOMINAL TERATOMA IN AN INFANT. MARTAGAO GESTEIRA, Pediat. e puercultura **3**:251 (Dec.) 1934.

A child of 20 months had become exceedingly dystrophic following frequent attacks of gastro-intestinal disorders. He was considered to have celiac disease because of an enlarged abdomen. He was examined under an anesthetic and was found to have a huge tumor, which was removed by operation. It was a teratoma and weighed 1,800 Gm., which was 20 per cent of the total weight of the infant. Recovery was uneventful.

SANFORD, Chicago.

GANGRENE FOLLOWING A SUBCUTANEOUS INJECTION OF DEXTROSE AND EPINEPHRINE. ALVARO BAHIA, Pediat. e puercultura **3**:265 (Dec.) 1934.

A child 1 month old was dehydrated following a gastro-intestinal disturbance. Dextrose and epinephrine hydrochloride were first given intraperitoneally, with no ill effect. They were then given subcutaneously. Five days later, gangrene of the extremities began, and the child died. The dextrose was given in a 5 per cent solution and the epinephrine in a 1 : 1,000 solution. The amount of dextrose given is not stated.

SANFORD, Chicago.

GONOCOCCAL ABSCESS OF THE MYOCARDIUM WITH MALIGNANT ENDOCARDITIS. FERNANDO R. RUÍZ, Rev. méd. latino-am. **20**:265 (Dec.) 1934.

Three patients with gonococcic abscess of the myocardium, which was diagnosed clinically, were studied at necropsy. The patients were men; the ages ranged between 30 and 37 years. In all the cases the abscess was in the membranous part of the interventricular septum. It opened first into the ventricular cavity, between the anterior and posterior aortic valves on the right. The gonococcus was isolated in all the cases.

DWYER, Kansas City, Mo.

ADDISON'S DISEASE TREATED BY PANIRRADIATION. NICOLAS CAPIZZANO, Rev. méd. latino-am. **20**:407 (Jan.) 1935.

Capizzano reports good results in Addison's disease by irradiation of the whole body with stimulating doses of the roentgen rays.

DWYER, Kansas City, Mo.

HEART BLOCK IN A CHILD FOLLOWING LOBAR PNEUMONIA. PAUL VON KISS and BÉLA WOLLEK, Arch. f. Kinderh. **104**:38, 1934.

A case of heart block with a ratio of 2:1 between the rate of the ventricle and that of the auricle in a 4 year old boy began two days after the crisis of lobar pneumonia. The radial pulse rate was 60 beats per minute. Treatment consisted of absolute rest in bed, 10 cc. of a 20 per cent solution of dextrose given intravenously daily and strychnine nitrate given subcutaneously. The heart block lasted eight days. Each day during the duration of the heart block an electrocardiogram was taken to follow the course of the condition.

LEMASTER, Evanston, Ill.

THE QUESTION OF FUNCTIONAL (ORTHOSTATIC) ALBUMINURIA. KARL DIETL and WALTER KOHLMAIER, Arch. f. Kinderh. **104**:80, 1935.

The various theories concerning the cause of functional albuminuria are discussed. The authors studied a series of their own patients with functional albuminuria and tried out various tests. In some cases application of cold to the region of the kidney caused albuminuria, presumably owing to a nervous reflex. Lordosis, particularly when the curvature was below the renal veins, seemed to be a factor in some children. At times assuming an exaggerated lumbar curvature caused albuminuria. The erect posture was found to produce albuminuria in other children. Lability of the vasomotor system is also considered to be a prominent factor in the etiology of functional albuminuria, as are mechanical and reflex causes.

LEMASTER, Evanston, Ill.

STORAGE DISEASES IN CHILDHOOD. WILHELM HERTZ, Arch. f. Kinderh. **104**:106, 1935.

In this *Uebersichtsreferat* Hertz discusses the present knowledge of diseases characterized by abnormal storage of fat and glycogen, such as Gaucher's disease, Niemann-Pick's disease and von Gierke's disease. LEMASTER, Evanston, Ill.

CUTANEOUS MANIFESTATIONS OF DISEASES OF THE LUNG IN CHILDREN AND THEIR RELATION TO DIAGNOSIS AND PROGNOSIS. E. MAYERHOFER and N. SKRIVANELI, Arch. f. Kinderh. **104**:137, 1935.

In many diseases of the chest in children, both tuberculous and nontuberculous, there is an involvement of the skin of the affected side of the thorax. Two distinct phases are observed: (*a*) In the beginning of an acute illness there is a marked thickening or swelling, and (*b*) in a longer chronic course there is often a thinning or atrophy of the skin. The transition from the first to the second phase does not always occur; its appearance is a sign of poor prognosis. The phase of swelling is an important diagnostic sign, calling for thorough search for a pathologic condition of the chest.

CRANE, Chicago.

A CASE OF AMYLOID KIDNEY WITH SUBACUTE COURSE SIMULATING HYPOCHLOREMIC AZOTEMIA. PAUL VON KISS and EDMUND KERPEL-FRONIUS, Arch. f. Kinderh. **104**:158, 1935.

A case is reported of a boy with pulmonary tuberculosis and nephrosis who within a short period after the onset of diarrhea showed marked hypochloremia and azotemia. The rapid loss of renal function in a typical case of nephrosis with normal blood pressure following diarrhea suggested hypochloremic azotemia, but the final clinical diagnosis of amyloidosis was confirmed at necropsy.

CRANE, Chicago.

WORM INFESTATION IN MECKLENBURG CHILDREN. H. BRÜNING, Arch. f. Kinderh. **104**:170, 1935.

Of 2,053 children examined, 17 per cent were found to harbor intestinal worms. The incidence was greater in the older children. The proportions in which the various types of worms were found varied with the age of the children and according to whether they lived in the city or the country. CRANE, Chicago.

FAMILIAL CIRRHOSIS OF THE LIVER. H. OPITZ, Jahrb. f. Kinderh. **144**:1 (Jan.) 1935.

A girl of 8 and her brother, aged 4, showed considerable enlargement of the liver and spleen. In the third year ascites developed. The body and mental development were normal. There was a tendency to cutaneous bleeding, bilirubinemia, urobilinuria, hypochromic anemia, leukopenia and thrombopenia. The diagnosis of cirrhosis of the liver was obvious. It is emphasized that it was difficult to tell whether the cirrhosis was the atrophic or the hypertrophic type and that this differentiation in childhood is difficult. In these two cases there was no noteworthy disturbance in the carbohydrate metabolism.

Splenectomy on the girl was followed by apparent noteworthy improvement. This operation was not done in the case of the boy.

The etiology was undetermined. Reference is made to numerous other cases of familial cirrhosis of the liver reported in the literature.

LANDAU, Rochester, N. Y.

TREATMENT OF MUSCULAR DYSTROPHIES IN CHILDHOOD WITH GLYCINE. OTTO BECK, Monatschr. f. Kinderh. **61**:296, 1935.

Beck studied the urinary nitrogen, creatine and creatinine in four children with progressive muscular dystrophy. In adults with muscular dystrophy varying amounts of creatine are excreted in the urine. When from 10 to 15 Gm. of glycine is added daily to the diet, the excretion of creatine increases markedly and after fourteen days drops to where it was before treatment, while the excretion of creatinine rises. Associated with these changes is clinical improvement.

In the four children studied the trends were somewhat as in the adult, but by no means as definite or as constant. Often creatinine could not be made to disappear from the urine.

The disappointing feature of the work is that with the atypical metabolism clinical symptoms showed no improvement.

Beck concludes that the treatment is a failure in children. In almost all other conditions preventive treatment in childhood tends to lessen morbidity in adult life. Unfortunately, in this disease the treatment is of value only after the condition has become established in adult life. GERSTLEY, Chicago.

THE TREATMENT OF PRURITIS ANI IN OXYURIS INFECTION. K. OXENIUS, München. med. Wchnschr. **81**:1977 (Dec. 20) 1934.

Oxenius uses ethyl aminobenzoate in the anal region and has had remarkable success in stopping the pruritis present in oxyuris infections. He has found it more effective than enemas with vinegar or garlic or the application of mercury ointment.

BRAHDY, Mount Vernon, N. Y.

A MESENTERIC CYST WITH THE STRUCTURE OF AN ESOPHAGUS. S. FLODERUS, Virchows Arch. f. path. Anat. **293**:608, 1934.

A mesenteric cyst removed from a girl aged 11 years had a length after fixation of 7 cm. and a diameter of 2.5 cm. Its lumen was lined in greater part by multilayered squamous epithelium like that of the esophagus. The epithelium rested

on a thick muscularis. In places the lining was like intestinal mucosa, with single-layered columnar epithelium and crypts of Lieberkühn. Groups of mucous glands were embedded in the wall. Floderus postulates an origin from pluripotent tissue separated from the entoderm in early embryonic life.

SCHULTZ, Evanston, Ill.

PURPURIC DISEASES IN CHILDREN. HEINRICH BAAR, Wien. klin. Wchnschr. **47**: 1039 (Aug. 24) 1934.

Heinrich Baar reports five cases of infantile purpura and discusses the importance of the functional thrombocyte test. Case 1, a typical case of purpura haemorrhagica (Werlhof's disease) occurred in an 8 year old girl. On examination the patient showed numerous irregularly distributed petechiae, ecchymoses and larger suffusions and punctiform and streaky mucosal hemorrhages of the hard and soft palate; the Rumpel-Leede phenomenon was markedly positive but was confined to the flexor surface of the elbow joints; thrombopenia was present, the platelets numbering 19,000; the bleeding time was fifteen minutes; the coagulation time was normal; retraction of the clot was absent. An important observation was that high thrombocyte counts were found only when the platelets were counted in a chamber, according to a method indicated by Baar, but not in ordinary smears (Boshammer, Hoffmann-Floessner) or in magnesium sulfate smears according to the method of Fonio. The functional thrombocyte curve was like that seen in cases of symptomatic thrombopenia. After the splenectomy it was temporarily almost normal; then it again became pathologic and variable, but on the whole it was improved.

Cases 2 and 3 were instances of purpura of the Schönlein-Henoch type (purpura rheumatica and purpura nervosa). They show the practical importance of the question of invagination. Case 2 was that of a 3½ year old boy; articular pains and itching eruption had been present for the past few months; numerous petechiae occurred on the flexor and extensor surfaces of the extremities and on the trunk; the abdomen was slightly sensitive to pressure and somewhat tense; the blood picture showed leukocytosis, with a marked shift to the left but nothing pathologic; examination of the urine showed acetonuria. On the day after admission to the hospital vomiting and severe collapse occurred. Although the thought of purpura rheumatica, with invagination as a frequent complication, had arisen, an operation was postponed because there was no blood in the stools and no palpable tumor was present. Autopsy revealed an ileac invagination with a submucosal hematoma in the invaginated section. Baar concludes that it is better to perform a laparotomy even before it is evident that invagination is present, at the risk of its proving superfluous, than to wait until it is too late.

Case 4 was one of anaphylactoid purpura occurring in a 10 month old girl. One extraordinary feature about this case was the youth of the patient; another, the marked exudative character of the changes, which distinguishes the anaphylactoid purpura from the Werlhof type of the disease.

ABT, Chicago.

TREATMENT OF DISEASES OF THE THYROID GLAND IN CHILDHOOD. EDMUND NOBEL, Wien. klin. Wchnschr. **48**:55 (Jan. 11) 1935.

Two groups of pathologic conditions must be distinguished, according to Nobel: those which affect the thyroid gland directly and those which lead to an alteration of the physiologic function of this organ. Whether or not the theory of iodine deficiency (de Quervain, Hunziker, Wagner-Jauregg) is justified, it is certain that iodine prophylaxis as practiced by Hunziker in Switzerland and Wagner-Jauregg in Austria is effective. If iodine therapy is necessary, it does not make much difference what preparation is used. Benign goiters run their course without provable disturbance of secretion and are to be sharply separated from those disease processes which produce a disturbance of the physiologic function of the thyroid gland. There are two groups: those in which there is diminished function of the thyroid and those in which there is increased function. The second group

is relatively rare in childhood. Nobel describes in detail a method for evaluating the amount of a thyroid preparation to be used in hypothyroidism. He also discusses the intellectual development of children suffering from hypothyroidism.

ABT, Chicago.

COMBINED TREATMENT OF ANEMIA IN CHILDREN. GERHARD HANTSCHMANN, Ztschr. f. Kinderh. **55**:51 (May) 1933.

Hantschmann reports the results of the treatment of anemia by a combination of iron, liver and blood transfusion. This form of therapy, which was used at the Children's University Clinic of Königsberg between 1929 and 1931, was more successful in combating the anemia than was the treatment with a single preparation which was used between 1925 and 1928. The combined treatment shortened the length of the disease and gave better end-results. In leukemia the improvement was only temporary, but in morbus Werlhofii it was more permanent.

LEVINSON, Chicago.

EOSINOPHILIA IN ALLERGIC CONDITIONS. GRIEBEL and DIMITROFF, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **25**:327, 1934.

Following a brief review of the literature, Griebel and Dimitroff describe their own clinical and experimental observations. They reach the conclusion that in the development of eosinophilia factors are active which are closely connected with ultraviolet radiation and with meteorologic changes. These factors exert their influence by way of the sympathetic nervous system. Clinical as well as experimental studies indicate that the chemical action of the rays on the body surface produces active substances which enter the blood stream and then the bone marrow and here effect a flooding out of the eosinophils. The authors assume that the melanin-like bodies which produce pigmentation are the eliciting cause, the building stones, of proteins such as tyrosine, tryptophan, proline and hydroxyproline which provide suitable materials for the formation of melanin. Although some investigators have assumed that histamine bodies play a part, the authors think that they do not, since neither histamine nor the related derivatives produce a reaction. Moreover there is the possibility that the entire reaction takes place by way of the sympathetic nervous system, for irradiation with ultraviolet rays increases the irritability of the sympathetic nervous system, and this would provide a common denominator for the meteorologic influences. Moreover, the problem of eosinophilia involves the reticulo-endothelial system. The spleen intervenes as a regulator of the blood in cases of an increased as well as of a decreased number of eosinophils. However, this aspect of eosinophilia still involves a number of unknown factors.

EDITOR'S ABSTRACT. [ARCH. OTOLARYNG.]

DIAGNOSIS AND TREATMENT OF CONGENITAL PYLORIC STENOSIS. J. POKORNÝ, Časop. lék. česk. **71**:1286 (Oct. 7) 1932.

The author reports twelve cases of this condition. He regards vomiting and palpable tumor (present in eleven cases) as the outstanding diagnostic points. Operation is recommended as soon as the diagnosis is made. Preoperative medical treatment is a contraindication, because it increases the emaciation of the patient. Three of the twelve patients died after operation.

STULIK, Chicago.

RENAL RICKETS. HARALD A. SALVESEN, Acta med. Scandinav. **83**:485 (Oct. 17) 1934.

In a 15 year old boy with chronic nephritis of long duration and symptoms of rickets for about one year complete studies of the acid-base balance of the serum were made before and during treatment with calcium lactate, with cod liver oil and with the two substances combined. The blood urea content was 368 mg.

per hundred cubic centimeters; the total base content and the calcum content of the blood were low; the potassium content and phosphate content were high; uncompensated acidosis was due to loss of base. Treatment with calcium alone or combined with viosterol resulted in marked improvement; uremia, pains due to rickets and acidosis disappeared; a rise in the calcium content and a fall in the phosphate content and potassium content of the blood took place. When ultraviolet irradiation was given alone symptoms of uremia and hypocalcemia and acidosis returned: the urea content and the mineral content of the blood varied inversely and equimolecularly. The boy died three months later, in a state of uremia in spite of the continued calcium therapy. Renal rickets is due to calcium deficiency resulting from retention of phosphate.

Parathyroid hyperfunction produced by the calcium deficiency contributes to the changes in the bones.

KAJDI, Baltimore.

ALUMINUM AS A FACTOR CONTRIBUTING TO THE RISE AND PROGRESS OF DIFFERENT PATHOLOGIC PROCESSES IN THE ORGANISM. A. L. TCHIJEVSKY and T. S. TCHIJEVSKAYA, Acta med. Scandinav. **83**:501 (Oct. 17) 1934.

The article gives no detailed data. The authors state that food cooked in aluminum vessels undergoes changes in taste, p_H and color in from two to ten hours; these changes do not occur in food prepared by similar methods in glass or enamel containers. After ingestion of food prepared in aluminum containers the feces of persons observed contained an increased amount of protein derived from the intestinal tract; this was regarded as evidence of inflammation. According to the authors colitis and occasionally nephritis or hepatic disease may be due to the use of aluminum pans, and the condition improves when glass vessels are used. After the patients got well and then ate a single meal which without their knowledge was prepared in aluminum utensils the colitis recurred in from one to two days. Spontaneous cancer developed forty times more frequently in white mice fed from aluminum containers than in those fed from other containers. The authors state that colloidal aluminum hydroxide, which is liberated from the utensils, carries a positive charge and may neutralize the negative membrane potential of the living cell.

KAJDI, Baltimore.

OSSIFICATIO PRAECOX. KNUD H. KRABBE, Hospitalstid (supp.) **77**:45 (Dec. 4) 1934.

A girl of 15 had always been well. Her menses began when she was just 11, and her breasts developed at the same time. She had always been chubby and became very fat from her ninth year.

She entered the hospital for treatment of obesity. General neurologic examination, blood tests, the hemoglobin content, urinalysis and roentgenograms of the cranium showed nothing abnormal. The basal metabolic rate was 94 per cent. The Aschheim-Zondek test and that for the follicle-stimulating hormone were negative. The serum calcium was 11.4 mg. per hundred cubic centimeters. The patient was markedly adipose; the weight was 77 Kg., and the height, 159.5 cm. Roentgenograms of the left wrist showed no sign of epiphyseal lines at the lower extremities of the radius and ulna, and the left elbow and ankle were devoid of epiphyseal lines (the normal age at closure is 20 years). Such early closure commonly indicates precocious puberty; nevertheless, the age of 11 is within the normal limit. Consequently, in this instance the potentiality of the ovarian hormone must be regarded as high, and the case is to be considered one of early ossification rather than one of precocious puberty.

VINTON, Brooklyn.

WELANDER'S ULCER. SVEN EMANUEL, Hospitalstid. (supp.) **77**:46 (Dec. 25) 1934.

A girl of 13 years had suffered for about three weeks from a genital ulcer which was very tender and smarted and was bathed in a watery secretion. The inguinal

glands were inflamed. Five times before she had had a similar sore, but it was quickly cured under treatment which now was ineffectual. Examination showed an oval ulcer the size of a quarter on the inner aspect of the right labium majus with sharp crimson edges, not undermined, and a slightly excavated base, moderately firm and covered by a greenish-yellow membrane. An abundant secretion without fetor was given off. The rim was slightly edematous, and the right inguinal glands were nodular. The temperature rose to 38 C. (101 F.). A similar ulcer of small size had existed eight days earlier on the opposite labium and small blebs on the nates. Under application of calcium chloride the original ulcer doubled in size, but it slowly healed when a solution of neoarsphenamine was applied locally and a single intravenous injection given. The Wassermann test and examination for spirochetes and Ducrey's bacillus all resulted negatively, as did the Pirquet test and examinations of the blood. In the detritus there was a rod-shaped bacterium differing from that described by Lipschütz in similar ulcers and also from Döderlein's vaginal bacillus and apparently justifying classification in a separate category.

VINTON, Brooklyn.

AGRANULOCYTOSIS AFTER AMINOPYRINE. P. STENBECK, Nord. med. tidskr. **9**:81 (Jan. 19) 1935.

Stenbeck reports ten cases of agranulocytosis, with two recoveries. All the patients were adult women who during long periods (months or years) had taken aminopyrine in a dose of from 0.4 to 0.9 Gm. a day as a remedy against rheumatoid pains.

WALLGREN, Gothenburg, Sweden.

CIRRHOSIS OF THE LIVER AND HYDRONEPHROSIS. S. VAN CREVELD, Maandschr. v. kindergeneesk. **3**:405 (Sept.) 1934.

In an infant aged 10 months cirrhosis of the liver and bilateral hydronephrosis occurred simultaneously. During life the child showed a particular disturbance in carbohydrate metabolism, principally characterized by the presence of a combination of glycosuria and acetonuria and a tendency to hypoglycemia; there was also marked excretion of glycuronates.

AUTHOR'S SUMMARY.

HYPERTHYREOSIS IN CHILDREN. T. HALBERTSMA, Maandschr. v. kindergeneesk. **3**:475 (Sept.) 1934.

Halbertsma discusses the occurrence of struma in Dutch children. In the Netherlands a greater frequency of struma and a low iodine content of the water run parallel. Both on clinical and on pathologic grounds it is desirable even in the case of children to separate exophthalmic goiter from other types of hyperthyreoses. A case observed in a girl aged 9 years is reported.

VAN CREVELD, Amsterdam, Netherlands.

A PARTICULAR CASE OF RELAXATIO DIAPHRAGMATIS. H. M. BROUWER-FROMMANN, Maandschr. v. kindergeneesk. **4**:129 (Jan.) 1935.

An infant of 4½ months fell ill with coughing and vomiting, and there were changes in the lungs. Roentgenograms showed relaxatio diaphragmatis, which is rare in childhood. The author discusses the differential diagnosis from phrenic paralysis caused by trauma at birth and hernia of the diaphragm. At the age of 9 months the child died of pneumonia. At the autopsy it appeared that the right part of the diaphragm was normal. Of the left half the front and lateral part was very thin and extended, forming a cupola in which parts of the stomach and of the colon and liver were present; the medial and back parts of the left half of the diaphragm were somewhat thicker than normal. The cause of this relaxation is discussed and a comparison is made with a case described by Fidler in 1914.

VAN CREVELD, Amsterdam, Netherlands.

Nervous Diseases

A New Treatment for Anterior Poliomyelitis and Encephalitis and Its Experimental Basis. N. D. Royle, M. J. Australia **1**:486 (April 20) 1935.

The author states that acute anterior poliomyelitis is an acute inflammatory disease and that the toxin of the disease, as in all acute inflammatory processes, constricts the arterioles and venules. This leads to acute dilatation of the capillaries, with stasis and consequent edema. The problems presented in the treatment of the disease are: (1) prevention or relief of the edema when it is present and (2) prevention of wasting of the involved muscles until the cells of the anterior horns of the spinal cord have recovered sufficiently to carry on their normal function again.

On the basis of the pharmacologic action of ephedrine in producing contraction of the capillaries, immediately followed by dilatation of the arterioles and venules, thus increasing the blood supply to the affected parts, an experiment was carried out with a goat to demonstrate this effect. The spinal cord of the goat was divided at the level of the third and fourth thoracic segments. The activity of the organ was successfully maintained by the frequent administration of small doses of ephedrine. The primary and temporary effect of ephedrine is hyperactivity of the cord with increase in the plastic and postural tone. This is dependent on the constriction of the capillaries and consequent temporary anoxemia.

In a second experiment it was demonstrated that wasting of denervated muscle groups could be prevented by the action of ephedrine. Six full-grown rabbits were used in this experiment, and the common peroneal nerve was divided on one side in each animal. Three rabbits were given ephedrine, and three were used as controls. At the end of thirty days the weight of the denervated muscles of the animals treated with ephedrine was about 13 per cent greater than that of the muscles of the controls.

The action of ephedrine in maintaining the activity of the spinal cord and in controlling the wasting of involved muscle groups following edema of the cord was demonstrated in four cases of poliomyelitis associated with paralysis. In these cases ephedrine was administered in doses of from 0.005 to 0.0075 Gm. from two to three times daily by mouth. In one case of especially serious involvement in a boy 4 years of age with almost complete paralysis of the extremities, the dosage was 0.0075 Gm. every three hours day and night. The ephedrine was administered for a period of four weeks in one case. The author concludes that small doses of ephedrine given frequently may prevent edema and subsequent paralysis if given in the preparalytic stage. If given in the postparalytic stage it prevents wasting of the involved muscles.

Gonce, Madison, Wis.

The Study of Early Infancy: A Plea for Cooperation Between the Sciences of Medicine, Psychology and Education. P. M. Bachelard, M. J. Australia **1**:554 (May 4) 1935.

"Character development may be likened to the formation of a statue by the pouring of metal into a mould," native endowment being the metal and the environment into which the child is born, the mold. The more one studies the development of character the more evident it becomes that the early years are of primary importance in determining the end-result. The character will develop in later years, but the pattern determining the lines of its development has been laid down early, under the influence of home, parents, relatives and companions. If one could get an accurate estimate of the child's inherent abilities and of the characteristics of his environmental "mold" during the first three years of life, one should be able to predict the type of adult that will result. Education at the so-called school age is so universally recognized as important that in many places it is compulsory, and yet by this time many of the character-forming traits are so firmly established that little corrective work can be done. It requires two or three years of school life before the teachers are able to say that a child is developing mentally at a subnormal rate.

Accurate methods of estimating the physical development of the person are available, and it is of prime importance to develop equally accurate methods of determining the rate of mental development in the earliest years. The establishment of child study stations connected with universities, such as are now being maintained at the University of Iowa and at Yale University, will result in much useful information, and it should not be feared that they will tend to turn out standardized persons. If one were able to describe mental retardation in the early years of life, there would be much better opportunities for medical and psychologic studies, and educators would be able to vary their methods of teaching to fit the capacities of the individual. That there will always be a large proportion of mentally defective persons seems inevitable, as the application of eugenic methods on a large scale is impracticable. Mental deficiency is generally regarded as incurable, but when one considers that the mental and physical picture of cretinism was, until recently, considered incurable, it becomes evident that great revision of the present ideas may be necessary.

It has been objected that it is wasteful of both energy and money to pursue educational methods with mentally defective persons. "Setting aside the opinion that physical, intellectual and economic efficiency should be the only aim of progressive states and that the devoted care given to the weaker members has not a moral and ennobling effect on the whole community, we cannot deny that in the long run it is cheaper to educate defective children to become contented, law-abiding and to some extent self-supporting citizens than to allow them to drift till they reach adult age and then to pay enormous sums for their support or incarceration."

TENNEY, Madison, Wis.

PHYSICOCHEMICAL STUDY OF THE BLOOD OF TEN ADULTS PRESENTING A CHVOSTEK SIGN. JACQUES DECOURT and CH.-O. GUILLAUMIN, Compt. rend. Soc. de biol. **115**:1048 (March 10) 1934.

On the blood of ten adults having either a one or a two plus Chvostek sign were determined the p_H, the alkaline reserve and the total, free and combined calcium, potassium and phosphorus contents.

In two cases in which tetany was present a calcium content of 6.4 and 8.8 mg. was shown, and in one in which marked vomiting occurred the alkaline reserve was 106. Otherwise, the results of the determinations were within normal limits, even though the patients were of nervous types, with disturbances of the vegetative nervous system.

WAHL, Cleveland.

ANATOMIC AND PATHOGENIC CONSIDERATIONS OF THE MYOPATHIES. R. POINSO and Y. POURSINES, Rev. franç. d'endocrinol. **12**:357 (Oct.) 1934.

Poinso and Poursines report a clinical and anatomic study of a case of the Leyden-Moebius type of myopathy and discuss the various pathogenic theories, especially the autonomic-endocrine. The case was that of a boy who began to have difficulty in walking at the age of 10. Six years later he was bedridden, with contractures in flexion in both lower extremities and the feet in the equinovarus position. Except for the right ankle jerk, all deep and superficial reflexes in the lower extremities were abolished. The motility in the upper extremities was greatly reduced, and there was atrophy of the left thenar group. The deep reflexes were abolished in both arms. The electrical reactions were decreased in all the affected muscle groups. There were no fibrillations or sensory changes. About three months prior to death hard masses appeared in the breasts and numerous small nodules under the skin of the abdomen, chest and neck. Coincident with this the patient began to lose weight rapidly. There was also enlargement of the liver and of the left testis. Autopsy was performed a few hours after death. The skeletal muscles were pale and reduced in volume. No trophic changes were present in the musculature of the heart and intestines. There was invasion of an epitheliomatous tumor mass into the pancreas, spleen, liver, right adrenal gland, testes, lungs and intestines. Histologic study of the striped musculature

revealed changes in the transverse striation, nuclear proliferation, formation of sclerotic tissue, sclerosis of the sarcolemma and of the interstitial tissue and collagenous metaplasia of muscle fibers. No young muscle cells or vacuoles could be seen in the preparations. The spinal cord showed changes in the cervical, lumbar and sacral segments. Some of the motor cells of the anterior horn were enlarged, and the basophilic substance was increased. In the intermediolateral process of the column of Clarke there were changes consisting of hypertrophy of the cells with displacements of the nucleus and transformation of the nuclei into a homogeneous substance which stained pale blue with toluidine. In addition, there were periependymal proliferation and thickening of the arachnoid. The rest of the central nervous system showed no evidence of change.

The authors review the various theories of pathogenesis. They think that the diminution and abolition of the tendon reflexes and the changes in the electrical reactions in their case speak in favor of an etiologic origin in the spinal cord, and in support of this they emphasize the pathologic changes in the spinal cord. They refer to the occasional combination of postencephalitic parkinsonism with the syndrome of myopathy as described by Tinel, Schiff and Courtois and to the degenerative changes in the striatal system and the subthalamic region as observed by Foix and Nicolesco. Then they discuss the theory of primary muscular involvement as advanced by Charcot, Landouzy, Dejerine and others. Finally, the authors discuss the endocrine theory postulated by Claude, Langeron, Dragonesco and others, who emphasized the combinations of myopathy with hypertrophy of the thyroid gland, hyperpituitarism or hypopituitarism and adrenal insufficiency. As they noted definite changes in the intermediolateral process of the column of Clarke in their case and as this structure has vegetative autonomic functions, the authors believe that their observation substantiates the vegetative-endocrine pathogenic theory of myopathy.

NOTKIN, Poughkeepsie, N. Y. [ARCH. NEUROL. & PSYCHIAT.]

LATE RESULTS OF TREATMENT OF SPASMODIC PARALYSIS IN INFANTS. MARCEL FÈVRE, Rev. d'orthop. **22**:220 (May) 1935.

The results of treatment in this class of patients are hard to estimate. The basis of judgment employed in this article was the ability to use the affected limb after treatment, as compared with that prior to treatment.

Tenotomy of the achilles tendon produced good results in ten of fourteen cases. In cases of involvement of the knee joint tenotomy alone or associated with the Stoffel operation or the Stoffel operation alone was used. The Stoffel operation produced better results than tenotomy. Scissor gait was treated with tenotomy of the adductor muscles and Stoffel's operation. When rotation was involved, osteotomy was undertaken.

Results depend on three factors: the intelligence of the child, the patience of the parents and the severity of the lesions.

In cases of hemiplegia fusion of the astragalus and cuboid bone with blocking of the ankle joint was effected.

RYAN, Vancouver, Canada.

PNEUMOCOCCIC MENINGITIS IN THE CHILD. A. CARRAU, C. PELFORT and N. LEONE BLOISE, Arch. de pediat. d. Uruguay **6**:121 (April) 1935.

Among eight hundred and fifty-nine cases of various forms of meningitis the authors found thirty-four in which the disease was due exclusively to the pneumococcus and thirteen in which the meningococcus, streptococcus or influenza bacillus was associated with the pneumococcus.

Most of the patients were past the first year of life. The mortality is practically 100 per cent. No form of treatment seems to have any effect. The few patients who have survived were treated with specific serum or quinine or with a combination of the two. Frequent lumbar punctures are always beneficial. The earliest possible diagnosis offers some hope for a more favorable effect of the therapeutic measures.

SCHLUTZ, Chicago.

MONGOLIAN IDIOCY IN BRAZIL. J. ROBALINHO CAVALCANTI, J. de pediat., Rio de Janeiro **2**:213 (May) 1935.

The author does not attempt to estimate the number of cases of mongolism in Brazil, but in the examination of one hundred and twenty-eight mentally deficient children he found that eighty-three, or 64 per cent, were mongolian idiots. He believes that most of the patients with mongolism showed a mixture of Negro blood, but he admits that it is difficult to draw any conclusions when the races are so intermingled.

SANFORD, Chicago.

PSYCHIC "ETHISMS." FRANZ HAMBURGER, Arch. f. Kinderh. **104**:92, 1935.

Hamburger suggests the word "ethism" for scientific usage in place of "habits," because of the misconception of the laity concerning the latter term. As ethismatic manifestations Hamburger includes nocturnal eneuresis, pavor nocturnus, chronic vomiting, polyuria, headache, tics, neurotic manifestations and psychic conditions, such as compulsory ideas, anxiety states, phobias, etc. All these conditions are considered to be based on an ethismatic etiology. In treatment suggestion, change of environment and training produce good results when properly carried out.

CRANE, Chicago.

EARLY INFANTILE PROGRESSIVE SPINAL MUSCULAR ATROPHY (WERDNIG-HOFFMANN) AND ITS RELATIONSHIP TO MYOTONIA CONGENITA (OPPENHEIM). OTTO SCHILDKNECHT, Deutsche Ztschr. f. Nervenh. **134**:163 (July) 1934.

Many authorities consider progressive spinal muscular atrophy and myotonia congenita as one and the same disease; others, Bielschowsky among them, hold them to be two distinct entities. Schildknecht reports five clinical cases and one with histologic observations with a view to throwing light on the subject. All five clinical cases were characterized by a typical constant progression of symptoms. Schildknecht considers this the most important diagnostic demarcation of Werdnig-Hoffmann's disease from myotonia congenita. In three cases there was a history of heredity, a point which he considers of diagnostic importance in Werdnig-Hoffmann's disease, although the succeeding children born to the parents may be normal. From his own observations, Schildknecht is unable to substantiate the finding that the disease is first manifested in the second half of the first year of life. He observed symptoms as early as the third week. Fibrillary twitchings were present in four cases but were limited to the tongue. The electrical reactions were normal, or there was a partial reaction of degeneration.

In one case of Werdnig-Hoffmann's disease Schildknecht observed regressive changes in the motor end-plates with gradual disappearance of the nerve elements. Comparing the anatomic changes in his case with those observed in cases of Oppenheim's disease, he noted certain similarities as well as some differences. In Oppenheim's disease the changes in the precentral convolution did not appear to be marked. Bielschowsky observed the pyramidal cells to be smaller, with abnormal dendritic formation. In his case of Werdnig-Hoffmann's disease Schildknecht noted marked diffuse changes in the ganglion cells. Neuronophagia was occasionally observed. The involvement of the cord was about the same in the two diseases. In neither were there inflammatory changes or circulatory disturbances.

The author found the peripheral nerves more markedly involved in Werdnig-Hoffmann's disease than in Oppenheim's disease. Schildknecht regards the change in the motor end-plates as a deciding factor in the differential diagnosis between the two. He saw well developed end-plates as well as some which were badly damaged in a number of atrophic muscle fibers. This can be explained only on the basis that originally all the motor end-plates were well developed and that the regenerative changes took place some time later.

Schildknecht also noted morphologic differences between the two diseases. Bielschowsky observed a splitting of the finer terminal nerve branches, and the fibrillar net was entirely missing. Schildknecht found the end-plates well formed, and the nerve net showed all types of transition from normal to those containing only a few granules. He concludes that the changes in the end-plates begin in the center and spread to the periphery.

In Oppenheim's disease, therefore, the primary pathologic change is in the end-plates; in Werdnig-Hoffmann's disease the end-plates are primarily normal and only later undergo degeneration. This leads to the conclusion that the changes in the anterior horn cells of the cord differ in the two diseases, although anatomically the end process is the same. It is inconceivable that in Werdnig-Hoffmann's disease the cells could have been pathologic from the beginning. Under such conditions normal end-plates could not be developed. Likewise it cannot be assumed that in Oppenheim's disease the degeneration follows a normal condition.

The pyramidal cells in the precentral area in Werdnig-Hoffmann's disease are more severely involved than in myotonia congenita. The changes in the muscles in both diseases are secondary, as a result of the destruction of the peripheral motor neuron, and especially because of the incomplete contact between the muscle and the nervous system.

From his observations Schildknecht considers that Oppenheim's and Werdnig-Hoffmann's diseases are distinct entities. The difference in prognosis of the two conditions thus becomes intelligible. In Oppenheim's disease there is incomplete development, which is capable of restitution and clinical improvement. In Werdnig-Hoffmann's disease a degenerative alteration is the primary factor. Progression of the degeneration is expressed in progression of paralysis. It is evident also that not in all cases will myotonia show improvement.

BERNIS, Rochester, N. Y. [ARCH. NEUROL. & PSYCHIAT.]

RESIDUAL PSYCHIC PHENOMENA FOLLOWING TRAUMATIC LESIONS OF THE HEAD IN CHILDREN. G. SSOUHAREVA, Ztschr. f. Kinderpsychiat. **2**:8 (April) 1935.

In seventeen cases of severe posttraumatic mental disturbance encountered and observed shortly after the initial injury there were definite symptoms of involvement of the brain (vomiting, convulsions and loss of orientation). In eight cases of milder disturbance a history of slight injury to the head was given. In the latter group four children had nervous complications with their psychiatric problems.

In analyzing these injuries of the head the author states that mild trauma is followed clinically by excitability, fear, confusion, impairment of effort, vague headaches, vertigo, impairment of memory and terrors at night. With psychotherapy these symptoms clear slowly, the impairment of effort being the latest symptom to disappear. The severe injuries manifest more serious symptoms, and these are of longer duration. Three syndromes are enumerated which may exist singly or collectively: (1) persistent intellectual impairment *(démence traumatique)*, (2) pronounced defect in the affective life of the patient (personality changes) and (3) epilepsy or epileptoid seizures.

The majority of the patients studied ranged in age from 7 to 11 years. Three children with contusion were from 1 to 3 years old. Generally, on the basis of the analysis of data in this study and of cases cited in the literature, the prognosis is gravest in persons in the lower age groups. The initial manifestations immediately following the injury of the head are not always an index to the ultimate severity of the condition, nor is one justified, except after prolonged study of the psychopathic results, in giving a definite opinion as to the final outcome. There are too many factors involved in the prognosis—exogenous, endogenous and psychogenic, each playing a rôle individually and in combination. The *"stigmates de la commotion"* must be viewed in toto.

JAHR, Omaha.

MUSCLE COMPENSATION IN INFANTILE SPINAL PARALYSIS. B. FREJKA, Bratisl. lekár. listy **15**:221 (March) 1935.

The author describes the case of an 11 year old girl who, in spite of complete loss of the deltoid, supraspinatus, infraspinatus, biceps and subscapularis muscles, was able to move the paralyzed upper limb. By contracting the triceps and the pectoralis major muscles so as to draw on the head of the humerus, she produced a subcoracoid luxation, after which movements of the forearm and hand could be effected. The paralysis was due to poliomyelitis. VINTON, Brooklyn.

CEREBRO-SPINAL MENINGITIS. C. F. BROWN and R. A. BROADY, Chinese M. J. **49**:155 (Feb.) 1935.

The authors report an epidemic of cerebrospinal meningitis in South Hunan, China, in the spring of 1934. During the preceding twelve years there had been a total of ten cases, all of which were sporadic. The epidemic came from Chenchow, 100 miles to the south, the first case being in a 3 month old infant. There were forty-three cases, in twenty-two of which complete recovery occurred. Seven of thirty-seven patients treated with serum, and all of the six not treated with serum, died.

The onset usually was so definite that it could be traced to a given hour in the twenty-four. In the cases of persons not receiving treatment which were reported in the city, death occurred within a few hours.

Ten cases were reported in which weak or questionable Kernig and Brudzinski signs were observed, all in children.

Three children in whom the condition was diagnosed and treated within a few hours of onset were up and well within one week. There were probably ten times as many unreported as reported cases in the city. Superstition prevented the institution of diagnostic and therapeutic procedures in most instances. Serum could not always be obtained early or in sufficient quantities.

NEFF, Kansas City, Mo.

A CASE OF CHOREA. M. P. YOUNG and Y. H. CHANG, Chinese M. J. **49**:260, 1935.

In a girl, aged 11, occurred the only case of chorea in the Elizabeth Blake Hospital, Soochow, China, in eighteen years. This patient had a mild attack, without fever and with a short relapse. She was discharged well after hospitalization for nearly three months. The report is warranted in China because of the extreme rarity of chorea there. NEFF, Kansas City, Mo.

PATHOTHERMIA. CORNELIA DE LANGE, Acta pædiat. **17**:83, 1934.

In 1929 de Lange reported the case of an infant who exhibited persistent fever, the cause of which was observed at autopsy to reside in multiple congenital anomalies and traumatic distortion of the central nervous system. The principal pathologic changes were located in the corpora striata and in the thalamus. The same author reported a similar case in 1933. In this patient the persistence of fever was explained by the presence of a ganglioneuroma of the spinal cord, which had originated in the thoracic region and had spread to involve the cervical portion of the cord.

In the present communication de Lange describes a case of complete transection of the spinal cord through the lower part of the cervical region. The patient was born in breech presentation, and delivery was effected by Mauriceau's maneuver. Transection of the cord resulted from necrosis due to concussion; the integrity of the vertebral column and of the meninges was not affected. There was no evidence of hemorrhage in either the spinal cord or the brain. Pathothermia was explained on the basis of the transverse lesion in the cervical cord. Histologic evidence indicated that the hypothalamic region was in the process of normal

development. "The clinical aspects of the case demonstrate that at this age the spinal cord possesses a high degree of autonomy and a considerable independence of the higher centers."

McCUNE, New York.

TREATMENT OF HEMORRHAGIC DIATHESIS, MORE ESPECIALLY ESSENTIAL THROMBOPENIA, WITH VITAMIN C. H. ENGELKES, Nederl. tijdschr. v. geneesk. **79**:679 (Feb. 16) 1935.

The author recommends the treatment of this disease with intravenous injections of vitamin C preparations and describes a case of morbus maculosus Werlhofii in a woman of 64, in whom two days after the injection of cevitamic acid the hemorrhagic condition disappeared.

VAN CREVELD, Amsterdam, Netherlands.

DISTURBANCES IN THE DEVELOPMENTAL FUNCTIONS OF GERM CELLS: A CASE OF ANENCEPHALY. V. W. D. SCHENK, Nederl. tijdschr. v. geneesk. **79**:767 (Feb. 23) 1935.

A case of a fetus of 7 months with acrania, rachischisis cervicalis, spina bifida sacralis and hernia ventralis is described. The nature of the deviations and the way in which the spinal cord can be duplicated are discussed.

VAN CREVELD, Amsterdam, Netherlands.

A FAMILY WITH MYOTONIA CONGENITA (THOMSEN'S DISEASE). J. SANDERS, Nederl. tijdschr. v. geneesk. **79**:782 (Feb. 23) 1935.

The author gives a description of a Dutch family of one hundred and thirty-three persons, seventy-four of whom had myotonia congenita (Thomsen's disease). He concludes that myotonia congenita is a simple dominant hereditary disease, occurring to the same degree in both sexes, and must not be considered to be a degenerative disease. No correlation proved to exist between myotonia congenita and mental disease.

VAN CREVELD, Amsterdam, Netherlands.

TYPUS DEGENERATIVUS AMSTELODAMENSIS (DE LANGE). R. VEDDER, Nederl. tijdschr. v. geneesk. **79**:993 (March 9) 1935.

A case of oligophrenia is described in a girl of 10 years, accompanied by the following symptoms: disproportionate nanosoma, facies typical of the disease, small crooked little finger and syndactylia. In 1933 two cases of the same type in children were described for the first time by Prof. Cornelia de Lange.

VAN CREVELD, Amsterdam, Netherlands.

CASE OF CEREBELLAR HEMORRHAGE IN A BOY OF 14 YEARS. S. M. LAMMERTS VAN BUEREN, Nederl. tijdschr. v. geneesk. **79**:4171 (Aug. 31) 1935.

All the causes commonly giving rise to cerebral hemorrhage in children failed to account for the symptoms in this boy during life. After death, which followed twenty-four hours after the beginning of unconsciousness, hemorrhage of the left part of the cerebellum was observed, where also some of the blood vessels appeared to be unusually enlarged.

VAN CREVELD, Amsterdam, Netherlands.

Diseases of the Eye

OPHTHALMOMYIASIS SUBRETINALIS. M. C. ENNEMA, Arch. Ophth. **12**:180 (Aug.) 1934.

The left eye of a 3 year old child was removed because of the presence of a tumor mass which projected into the vitreous. Sections of the globe revealed a marked inflammatory reaction with eosinophilia, which is rare in an eye. A structure not of human origin was found, which proved to be a Diptera larva.

WESCOTT, Chicago.

MUSCLE IMBALANCE IN MYOPIA. F. W. MARLOW, Arch. Ophth. **13**:584 (April) 1935.

Marlow states the belief that muscle imbalance is present in a high percentage of cases of axial myopia and that this imbalance is probably the most important active factor in the onset and progress of this condition. WESCOTT, Chicago.

LOCAL QUININE THERAPY IN CASES OF INTERSTITIAL KERATITIS AND OLD CORNEAL OPACITIES. ELIAS SELINGER, Arch. Ophth. **13**:829 (May) 1935.

Experiments on animals and clinical trials in cases of trachoma showed that 2 per cent or 4 per cent quinine bisulfate ointment or a 10 per cent aqueous solution can be applied locally daily for many months without any harmful effects on the conjunctiva or cornea. While the course of interstitial keratitis cannot be influenced by antisyphilitic or local therapy, both must be thoroughly employed. In three cases of bilateral active interstitial keratitis 2 per cent quinine bisulfate ointment was prescribed for use twice a day in addition to the local and general treatment. Rapid subjective improvement was noted in a few days, with lessening of the photophobia and blepharospasm. The appearance of the corneal opacities changed also. WESCOTT, Chicago.

SOME NOTES ON THE TREATMENT OF STRABISMUS. S. R. GIFFORD, Brit. J. Ophth. **19**:148 (March) 1935.

The object of the paper is to outline a routine of treatment which in the hands of Gifford and of the group working in his clinic has proved very satisfactory. Some of the practical points to be culled from the article follow: In esotropia with less than 1 diopter of hyperopia bifocal lenses or grab fronts of 2 diopters plus may be of service when the eyes are straight for distance but show from 10 to 15 degrees of convergence for reading. In concomitant squint of all types and degrees, when refraction was done early, usually before the age of 6, with no other treatment than the use of atropine or occlusion, it was found that after six months the eyes were straight with glasses in 12.5 per cent of the cases, and after eighteen months, in 30 per cent. In divergent squint little is to be expected from correcting the refractive error, as a rule. Attempts should be made to improve amblyopia and fusion.

The results with amblyopia have been disappointing. With occlusion and the use of atropine the results have not been remarkable. Prolonged occlusion when vision is below 6/60 is, as a rule, impossible. The use of atropine and a system of reducing vision in the good eye are of value in overcoming suppression of the poorer eye and as an aid to orthoptic training, but actual improvement in vision has not been striking.

In accommodative squint fusion training seems to be of value in hastening a cure but is not always necessary.

Squints due to defective fusion include conditions in which vision is good and there is little or no refractive error, many divergent squints, especially those in which the divergence is intermittent or latent, and alternating squints. In the latter group, while fusing ability is undeveloped, it cannot be said to be absent.

In squint with amblyopia, when vision is less than 6/60 or 6/24, fusion training is of little value. Attempts should be made to improve the amblyopia, and the effect of glasses is watched for from six months to one year. After this time surgical intervention is indicated. Squint with anisometropia is considered a variety of this group. Iseikonic glasses may bring about binocular vision if it does not exist with ordinary lenses.

In squint due to muscular abnormalities surgical operation is indicated without preliminary training.

Dividing all types of squint into those of high and those of low degree, Guibor found in forty-eight patients that when the angle of squint was 15 degrees or

less the eyes of 84 per cent became straight after refraction and orthoptic training, while when the angle was more than 15 degrees the eyes of only 16 per cent became straight.

The method of training is outlined. For overcoming suppression the large synoptophore of Maddox is ideal. The types of fusion charts are discussed.

Gifford believes that when progress is at a standstill operation should be advised regardless of age. Simultaneous operation on both lateral rectus muscles is adopted in nearly all cases of concomitant squint higher than 20 degrees. The eye with the poorer vision is selected. With advancement and recession, squint of 35 degrees and sometimes 45 degrees may be corrected by one operation. In the treatment of slight squint and phorias the O'Connor operation seems to have a definite place.

The article is illustrated. ZENTMAYER, Philadelphia. [ARCH. OPHTH.]

HYALINE BODIES ON THE DISCS. F. C. PLUMMER, Brit. J. Ophth. **19**:215 (April) 1935.

The interesting feature of the case was the extent and prominence of the hyaline bodies, which covered the surface and overlapped the margins of the disk in both eyes.

The patient was a boy, aged 10 years. The error of refraction was +2.5 in each eye. Vision was 6/6. ZENTMAYER, Philadelphia. [ARCH. OPHTH.]

XEROSIS IN THE EYE CLINIC. A NEW LOCAL TREATMENT. E. CORNET, Ann. d'ocul. **171**:484, 1934.

Xerosis, or xerophthalmia, or keratomalacia, is due to absence of vitamin A or, more exactly, to "avitasterinose A." It was not until 1902 and 1903 that Mackenzie and Koun noted the relation which exists between this disease and the state of nutrition.

Xerosis is found especially in the Orient. It often results in blindness. It may be cured by local treatment, but the prognosis remains serious because of the relation of the disease to the patient's general condition. The treatment suggested by Cornet is as follows: the subconjunctival injection of milk freshly sterilized at 115 F., in amounts of from 0.5 to 1 cc., into the eye every second, third, fourth and fifth day; the instillation of olive oil or cod liver oil; a prescription of milk, either human milk or that from fresh healthy cows or boiled milk, yolks of eggs, green vegetables, oranges and cod liver oil; the administration of the serum of Quinton and appropriate pulmonary and intestinal examination and treatment.

In case of a xerotic ulcer, the instillation of olive oil and cod liver oil should be omitted. Atropine or physostigmine, plus mild protein silver, may be used as indicated. MCKEE, Montreal, Canada. [ARCH. OPHTH.]

OCULAR MANIFESTATION IN THE EXUDATIVE DIATHESIS. H. BRACCI-TORSI, Ann. di ottal. e clin. ocul. **63**:359 (May) 1935.

This condition, which is common in children, was described by Czerny as a tendency to abnormal reactions of the skin, mucous membranes and lymphatic tissues to irritants which produce no effect in normal persons. He found that it was favored by excessive feeding and especially by excessive feeding of fats. Later work showing retention of sodium chloride in the skin of such patients has modified but not materially altered Czerny's conception.

Bracci-Torsi examined a series of infants showing persistent thymus, eczema, enlarged tonsils, adenoids and enlarged cervical glands with regard to the visual apparatus. Blepharitis and folliculosis were common, and in a number of the infants phlyctenulosis was found. There was nothing characteristic about these conditions which would distinguish them from similar lesions occurring in other-

wise normal persons. The response to treatment is suggested as a valuable aid in diagnosis, since lesions due to the exudative diathesis clear up on proper diet.

GIFFORD, Chicago. [ARCH. OPHTH.]

THE SO-CALLED COLOBOMA OF THE MACULA LUTEA. K. T. A. HALBERTSMA, Nederl. tijdschr. v. geneesk. **79**:1233 (March 23) 1935.

The author gives the results of observation in five cases of coloboma of the macula, in three of which the condition was unilateral and in two bilateral. In all the cases, both in the patients themselves and in most of their relatives other congenital ocular abnormalities were also found, mostly of mesodermal origin. From further observations the deductions were made that in cases of this abnormality a bilaterally preformed defect is present, the result of a fault in mesodermal development, which is, in connection with other observed congenital ocular anomalies, taken as a modification.

VAN CREVELD, Amsterdam, Netherlands.

A CASE OF LENTICONUS POSTERIOR. R. G. POSTHUMUS, Nederl. tijdschr. v. geneesk. **79**:1718 (April 13) 1935.

This condition was observed in a girl of 12 years. It is due to a coincidence of multiple factors which create the conditions for the formation of a hernia of the growing, soft lens substance, a hernial ring being produced by the more resistant, a hernial pouch by the weaker, portion of the limiting surface. Lenticonus may thus be assumed to be a hernia lentis, possibly a hernia lentis congenitalis.

VAN CREVELD, Amsterdam, Netherlands.

Diseases of the Ear, Nose and Throat

THE FLOOR OF THE MAXILLARY ANTRUM. J. W. GERRIE, J. Am. Dent. A. **22**:731 (May) 1935.

In a comprehensive article dealing with the anatomy and pathology of the antrum Gerrie includes the following among his conclusions: Cases of pathologic conditions in the upper alveolar process contribute approximately 20 per cent of the cases of maxillary sinusitis in patients applying for treatment. Alveolar abscess is seldom a demonstrable cause of sinusitis. Free socket dressings are dangerous in the presence of dental fistulas. Sinusitis following maxillary fracture is usually chronic. Soft organic foreign bodies more frequently cause acute suppurative sinusitis, while metallic ones are more commonly of a chronic irritating character. Gerrie believes that oral fistulas should not be used as a route for sinus irrigation. Reports of eight cases are included.

NOYES, Chicago.

VITAMINS IN THE TREATMENT OF ECZEMA. M. COMEL, Gior. ital. di dermat. e sif. **76**:283 (April) 1935.

Comel confirms his previous report on the beneficial effects of the administration of vitamin D in cases of eczema. He also states that the lactic acid content of the blood decreases as soon as therapy with vitamin D is instituted.

PARDO-CASTELLO, Havana, Cuba. [ARCH. DERMAT. & SYPH.]

A FEW CASES OF INFREQUENT NONDIPHTHERITIC STENOSIS OF THE RESPIRATORY TRACT. HERMANN ZISCHINSKY, Jahrb. f. Kinderh. **144**:191, 1935.

Three cases of primary phlegmonous laryngitis are described. One was in a child of 7 years, another in a child of 19 months, and another in a child of 3½ years of age. All the children had noisy respiration, stridor and high fever. The

inflammation subsided quickly, and the children were discharged cured following tracheotomy. In addition to these three cases, another one, in a child aged 2 years, which terminated fatally, is cited. The literature is reviewed, and the high mortality is stressed. A swollen epiglottis in the presence of the symptoms just described is suggestive of this condition.

A case of chronic hypertrophic laryngitis (verified by postmortem examination) in a mongolian idiot of 4½ years is described. Death followed the onset of symptoms comparatively suddenly. The early symptoms were undoubtedly unnoticed because of the low grade intelligence of the patient. Another case included in this category is that of a boy, 1 year of age, in which the early sign was a cough without laryngeal symptoms. Laryngeal symptoms appeared later. The duration of the illness was twelve weeks. Tracheotomy was necessary. Apparently a complete recovery followed.

Another striking case was that of a girl, 1½ years of age, who was ill for fourteen days. Symptoms of laryngeal stenosis appeared, which led to the thought of diphtheria. A positive culture tended to confirm this suspicion. The course was rapidly down-hill, and autopsy revealed sepsis with mycotic aneurysm of the aorta, which compressed the left main bronchus and involved the left recurrent nerve.

The last case reported is that of a 4 year old girl with symptoms suggesting a foreign body, subglottic laryngitis or compression of the trachea due to glands. A roentgenogram showed tracheal compression due to two small objects in the immediate vicinity. The symptoms subsided, and within eight days the child was ready for discharge from the hospital. The etiologic factor was not determined, but the course suggested an infectious process. LANDAU, Rochester, N. Y.

SYMPTOMATOLOGY AND CLINICAL ASPECTS OF OTITIS CAUSED BY FRIEDLÄNDER'S BACILLUS. F. ALTMANN, Monatschr. f. Ohrenh. **69**:513 (May) 1935.

Altmann points out that the reports of a number of investigators indicate that there is a connection between the clinical course of acute otitis and the type of micro-organism that is causal. Comparisons have been made between the otitides that are caused by noncapsulated and those that are caused by capsulated bacilli. Among the latter group, those caused by Streptococcus mucosus were given especial attention because of their relative frequency. However, later studies disclosed that the dependence of the clinical course on the type of the causal organism is not so great as was at first believed. A number of otitides were observed which had been caused by noncapsulated bacilli, such as hemolytic streptococci, but which took nevertheless a clinical course much like that observed in cases of infection with Str. mucosus, whereas Str. mucosus quite often caused otitis that took an ordinary course. To be sure, Wirth, who gave this problem his particular attention, found that 60 per cent of the otitides that are caused by Str. mucosus take the characteristic protracted course, whereas only 10 per cent of those caused by the ordinary streptococci do. According to recent bacteriologic studies, Str. mucosus is practically identical with Pneumococcus type III. In cases of infection with the other types of pneumococci (I, II and IV), however, the protracted course is comparatively rare. Reports in the literature indicate that there is considerable similarity between the course of the otitis caused by Str. mucosus and that of the otitis caused by capsulated bacilli of the Friedländer group; the latter, to be sure, is much less frequent. The small number of cases that had been reported and the fact that some authors stressed the great danger involved in this form of otitis induced Altmann to describe the cases that were observed in his clinic. He reports seven cases, in five of which the treatment was surgical and in two of which the condition healed spontaneously. In four cases the onset was insidious, without the characteristic clinical symptoms, the only signs being mild piercing pains in the ears, which disappeared again, and a reduction in the hearing capacity or tinnitus aurium. In three cases the onset was more acute. A paracentesis was never necessary. With the exception of one case, the disorder always took

an extremely protracted course. The onset was always followed by a comparatively long period of latency, during which only the impaired hearing capacity and the tinnitus aurium indicated the existence of a disorder. In some of the cases which terminated in spontaneous cure, the period of latency was followed by cure, while in the others signs of mastoiditis appeared. The indications for the surgical opening of the mastoid process were: in three cases, the appearance of a periosteal abscess; in one case, painfulness of the mastoid process on pressure, lowering of the posterior upper wall of the auditory meatus, profuse suppurating secretion, subfebrile temperatures and positive roentgenologic aspects, and in another case, slight painfulness of the mastoid process on pressure, lowering of the posterior upper wall of the auditory meatus, profuse suppurating secretion, occasional knocking in the ear and an increase in temperature. In these otitides caused by the Friedländer bacillus the cells of the mastoid process contained the peculiar whitish granulations that are characteristic for the otitides caused by Str. mucosus. Of the cases in which surgical treatment became necessary, only one presented an uncomplicated mastoiditis, while in the other four complications existed. One case terminated fatally, but the fatal outcome cannot be ascribed to the type of infection. Altmann agrees with Eigler, who maintained that the majority of otitides that are caused by the Friedländer bacillus are of rhinogenic origin and develop by way of the eustachian tube. In four of six patients who were examined for it, atrophic change of the mucous membrane of the nasopharynx could be detected. The author emphasizes that many underestimate the possibility of a spontaneous cure in otitis caused by the Friedländer bacillus. He states that Wirth observed three spontaneous cures and that he observed two.

EDITOR'S ABSTRACT. [ARCH. OTOLARYNG.]

OTITIS MEDIA IN NURSLINGS. A. TEN BOKKEL HUININK, Maandschr. v. kindergeneesk. **4**:219 (March) 1935.

Seven cases are described of children all of whom were suffering from an alimentary intoxication which could not be ameliorated. In all cases purulent otitis was present, and in all instances in which the antrums were opened pus was found. Otologic examination did not give strong indication for antrotomy. Nevertheless, this procedure was performed in some cases and resulted in immediate recovery.

VAN CREVELD, Amsterdam, Netherlands.

Diseases of the Skin

RINGWORM OF THE SCALP: CURABILITY, WITHOUT DEPILATING MEASURES, OF INFECTIONS CAUSED BY "ANIMAL" MICROSPORONS. GEORGE M. LEWIS, Am. J. M. Sc. **189**:364 (March) 1935.

Thirty-seven patients with tinea capitis were treated without depilating measures and with local applications only. The results were as follows: Uniformly good effects were obtained in thirteen cases in which the causal fungi were "animal" microsporons, and the condition was cured by local therapy alone in from three to twelve weeks; in twenty-four cases in which the condition was due to Microsporon Audouini, Achorion Schoenleinii and Trichophyton violaceum the disease failed to respond.

The ointment used consisted in the main of 1 per cent each of iodine crystals, thymol and oil of cinnamon in petrolatum. A different preparation was used in only four cases. Instructions were given to use the ointment twice daily, preceded by a shampoo of soap and water. The patients were reexamined as frequently as possible, and the clinical diagnosis was checked by the Wood light and microscopic and cultural determinations. Stress is laid on culturing for the causative fungus, the management of the disease being dependent on the findings.

HEUSKE, Omaha.

ESSENTIAL SHRINKAGE OF THE CONJUNCTIVA IN A CASE OF PROBABLE EPIDERMOLYSIS BULLOSA DYSTROPHICA. MARLIN COHEN and MARION SULZBERGER, Arch. Ophth. **13**:374 (March) 1935.

The authors report a case of lesions of the eye resembling those of pemphigus in a 7 year old boy having epidermolysis bullosa dystrophica. Biopsy of the skin showed changes characteristic of this cutaneous lesion. Neoarsphenamine seemed to bring about improvement. Traumatic bullae could be produced on two occasions. Potassium iodide seemed to cause exacerbation of the condition. From the appearance of the right eye a tentative diagnosis of phlyctenular keratoconjunctivitis was made. The tarsal conjunctiva was covered with a pseudomembrane and a posterior symblepharon occurred. The cornea became cloudy, and finally perforated, and the eye was enucleated.

WESCOTT, Chicago.

ECZEMA OF CHILDREN. SAMUEL LOUIS BAUER, West Virginia M. J. **31**:193 (May) 1935.

None of the existing classifications can well be applied to conditions occurring in early life, since in these cases lesions of different types may occur in the same patient.

The disease usually appears between the tenth week and the sixth month of life. From 3 to 7 per cent of infants in the United States suffer from some form of eczema. The etiology of eczema is perplexing. Hill expressed the belief that in about two thirds of all cases the cause is allergic. Other investigators have found a family history of allergy in from 70 to 80 per cent of the cases. The clinical diagnosis of infantile eczema often presents difficult problems. The treatment must consist of two kinds: local, palliative treatment and elimination of the underlying cause. The child with eczema must be protected from secondary infections.

In the search for causative sensitizations the cutaneous scratch tests are of great value. The use of intracutaneous tests is inadvisable in infants and young children. Milk and eggs are the chief offenders among foods, next to which are cereals, oranges and tomatoes. If a child is sensitive to cow's milk, he is probably sensitive to any kind of milk, even though reactions to the cutaneous tests are negative. When a patient is sensitive to a food, elimination of the food from the diet is the solution. Desensitization has not been successful. A child may be sensitive to more than one food or to a food only when in combination. For example, he may not be sensitive to either milk or wheat but may be definitely sensitive to milk and wheat in combination. A child may be sensitive to the bacterial flora of the nose, throat, skin or intestinal tract. In that case an autogenous vaccine may be given. While the present knowledge of the basic nature of eczema is defective, the existence of a state of allergy has been fairly well established.

SHAWKEY, Charleston, W. Va.

ERYTHEMA NODOSUM AND FOCAL INFECTION. L. BEDFORD ELWELL, Brit. M. J. **1**:974 (May 11) 1935.

A case is reported of recurrent erythema nodosum, apparently precipitated by axillary lymphadenitis. It is thought that both Mycobacterium tuberculosis and Streptococcus haemolyticus were ruled out as agents.

ROYSTER, University, Va.

SKIN DISEASES IN CHILDREN. R. T. BRAIN, Practitioner **134**:492 (April) 1935.

In an excellent article the author presents the conditions of the skin most frequently seen in the clinic for diseases of the skin at the Great Ormond Street Hospital. The conditions most frequently seen are eczema, impetigo, urticaria, scabies and seborrhea.

Many practical suggestions for the diagnosis and treatment of these conditions are given. Each subject is described in detail. Numerous prescriptions are given.

SAPPINGTON, San Francisco.

PROBABLE ACRODYNIA CHARACTERIZED BY A SYNDROME OF ABDOMINAL PAIN. JEAN LEVESQUE and R. MÉRY, Bull. Soc. de pédiat. de Paris **31**:528 (Dec.) 1933.

A boy, 4 years old, was suffering from abdominal pain. On physical examination the abdomen showed nothing abnormal, and the child seemed to be somewhat relieved when pressure was applied on palpation. He was miserable, and in some ways the condition suggested the diagnosis of acrodynia. The blood pressure was considerably elevated. He was given daily carbonated water baths and diathermy treatments, and in the course of a week he was free from symptoms, and the hypertension was no longer present. In another case of typical acrodynia of several months' duration a boy of 20 months recovered after similar treatment was instituted. It is thought that the vasoconstriction and hypertension are favorably affected by the action of carbonated water baths and diathermy on the sympathetic nervous system.

BENJAMIN, Montreal, Canada.

TREATMENT OF FURUNCULOSIS IN INFANCY. I. FISCHER, Orvosi hetil. **79**:156 (Feb. 9) 1935.

In case of severe furunculosis it is not advisable to open many furuncles at once, because this may cause a turn for the worse in the infant's condition. Therefore only matured furuncles, over which the skin has become thin, should be opened. The infants lie naked in a Dobszay hanging bed. The furuncles are treated with a concentrated solution of potassium permanganate or with a 2 per cent solution of acriflavine hydrochloride. Good results were obtained by the use of potassium soap baths, recommended by the child clinic in Düsseldorf.

GÖTTCHE, Budapest, Hungary.

Surgery and Orthopedics

CHRONIC APPENDICITIS AND CONGENITAL SYPHILIS IN THE CHILD. W. MIKULOWSKI, Arch. de méd. d. enf. **38**:147 (March) 1935.

The diagnosis of chronic appendicitis, especially in the very young, rests almost entirely on subjective symptoms of more or less value; it therefore lacks the elements of dependability. An objective feature, known as the muscular atrophy of Wolkowicz, is not confined to this disease but may be found in the multipara, in very obese subjects, in patients previously operated on and among laborers after retirement from active duty. Recently Buchmann of Rostow has added the objective sign of anisocoria. In from 88 to 93 per cent of the cases of chronic appendicitis in which the diagnosis was confirmed at operation there was dilatation of the right pupil, which slowly disappeared after surgical intervention. Buchmann asserts that through the intimate connections of the principal trunk of the sympathetic nervous system on the right side with the branches supplying the appendix there exists a possibility that excitants of any sort, mechanical or toxic, may exert their effect on the pupil of the same side.

Enriquez has called attention to the frequent affinity in the pathologic condition of the appendix, of the liver and of the gallbladder, giving rise to what he terms appendicular hepatitis or ascending lymphangiocolitis, accompanied by nausea, vertigo, constipation, pylorospasm or asthma. Secondary symptoms in these cases are attributed likewise to reflex vagotonic disturbances originating in appendicular inflammation.

Gibson stressed the great difficulty in arriving at a correct diagnosis among neurasthenics, regardless of age or sex. The supreme importance of eliminating

congenital infantile syphilis in these cases is noted, together with strong approval of mercury, which the author places beside quinine and antidiphtheric serum for dependable results.

AMESSE, Denver.

SPLENECTOMY IN GAUCHER'S DISEASE. L. DE CASTRO-FREIRE, Arch. de méd. d. enf. **38**:163 (March) 1935.

In 1926 the author reported the first case of splenectomy for Gaucher's disease in Portugal. The patient was a girl of 8 years suffering from splenomegaly, moderate hypertrophy of the liver, recurrent epistaxis and purpura. The operation, performed in 1923, was followed promptly by an amelioration of all symptoms. In 1934, eleven years after surgical intervention, opportunity was afforded to reexamine this subject, now a young woman of 19.

The history was satisfactory: No attack of purpura was reported in the entire postoperative period; epistaxis was noted only occasionally, and menstruation was fairly regular; osseous pains were absent, and the patient was able to meet the demands of hard manual labor. It may, therefore, be concluded with fairness that while splenectomy may not be dignified as a cure in these cases, there is an arrest of progress, and the procedure should be carefully considered in every case.

AMESSE, Denver.

ARTICULAR FOREIGN BODY IN THE ELBOW. R. BECQUET, J. d. sc. méd. de Lille **52**: 482 (June 17) 1934.

A boy of 12 years suffered a dislocation of the radius, which was promptly reduced, but pain and limitation of motion persisted for two months. Roentgenography showed nothing except a fragment at the tip of the trochlea. At operation the synovial border was found to be spreading so as to block the articulation. Histologic examination revealed tuberculous lesions. The condition was therefore tuberculous arthritis not previously recognized. Becquet discusses the origin of the condition and indicates the difficulty of diagnosis.

VINTON, Brooklyn.

DIAGNOSIS AND EARLY TREATMENT OF CONGENITAL DISLOCATION OF THE HIP. V. PUTTI, Pediatria **43**:1 (Jan.) 1935.

Putti presents a plea for early diagnosis in cases of congenital dislocation of the hip and illustrates with pictures his method of early treatment of this disorder.

He cites 200 cases in 94.9 per cent of which early treatment was productive of cure and points out that every patient should be submitted to this painless and effective management long before the age of 1 year; he emphasizes that the earlier the treatment is started the better the results.

SIGNORELLI, New Orleans.

ACUTE INTUSSUSCEPTION IN EARLY CHILDHOOD. D. MOGGI, Riv. di clin. pediat. **33**:72 (Jan.) 1935.

This paper is a discussion of acute intussusception in infants and young children. The importance of early diagnosis, the symptoms, the differential diagnosis and the treatment are considered.

HIGGINS, Boston.

DIAGNOSIS OF INTUSSUSCEPTION. A. LOURENCO JORGE, Arch. de pediat., Rio de Janeiro **7**:163 (Jan.) 1935.

The signs and symptoms of intestinal invagination are discussed.

SANFORD, Chicago.

USE OF SKELETAL TRACTION IN INFANTS; PRESENTATION OF A NEW USE. E. ROVIRALTA, But. Soc. catalana de pediat. **6**:233 (July-Aug.) 1933.

Skeletal traction is useful in children in treating fractures of the diaphysis of the femur, fractures of the lower end of the humerus and severe injuries, even without fractures, in which exposure without pressure is desirable.

WATSON, Elmhurst, Ill.

ACUTE INTESTINAL OBSTRUCTION IN INFANCY. CESAR IZZO, Rev. chilena de pediat. **6**:43 (Feb.) 1935.

The appearance of blood in the stool is the most important single sign in intestinal intussusception. As preoperative and postoperative measures, Izzo advises cardiac stimulants, the maintenance of adequate body heat, fairly large doses of atropine and gastric lavage before the operation. During the operation and immediately after, the infant must be guarded against chilling. As postoperative measures, the intravenous use of hypertonic solutions of sodium chloride and gastrointestinal lavage are advised. The author advises rapid hydration of the organism and early feeding.

SCHLUTZ, Chicago.

INGUINAL HERNIA IN THE CHILD. CARLOS URRUTIA, Rev. chilena de pediat. **6**:93 (March) 1935.

Surgical intervention is the best treatment for inguinal hernia. It is the only treatment in all strangulated forms, in hernias reduced with difficulty and in inguinal-scrotal forms.

The spontaneous disappearance of hernia is unlikely unless the hernia is very small. The successful outcome of the surgical procedure depends on the type of the hernia, the age of the child and the general state of its health.

SCHLUTZ, Chicago.

ANISOCORIA IN CHRONIC APPENDICITIS. K. SCHLAEPFER, Deutsche Ztschr. f. Chir. **244**:311, 1935.

Sawitzkij and Buchman first called attention to anisocoria in chronic appendicitis. This is due to changes which occur in the sympathetic plexus of an inflamed appendix during scar formation. The sympathetic fibers of the appendix belong to the superior mesenteric plexus. The fibers go through the solar plexus to the roots of the ninth and tenth thoracic segments, then through the spinal cord to the ciliospinal center, then to the carotid plexus and through the long ciliary nerve to the muscle of Müller and the dilator of the pupil. In one series of 2,000 cases Buchman found a relative dilatation of the right pupil in 88 per cent of the cases, and in another series of 1,500 cases, in 93 per cent of the cases. For studying the pupils a diffuse bright light is essential. The phenomenon does not occur in right-sided inflammation of the pelvic organs, the liver and gallbladder. Tertiary syphilis and right-sided pulmonary tuberculosis must be ruled out. In 83 per cent of 60 cases observed by the author in his series a relative dilatation of the right pupil was observed. The majority of these cases were those of acute exacerbation of chronic appendicitis. From two and one-half to five months after operation the patients with anisocoria showed equal pupils, in agreement with the observations of Sawitzkij and Buchman. The author believes that anisocoria is a useful accessory sign in the diagnosis of chronic appendicitis.

GLASER, Rochester, N. Y.

SURGICAL TREATMENT OF CONGENITAL PYLORIC STENOSIS AND ITS END-RESULTS. E. POLÁK, Časop. lék. česk. **71**:1281 (Oct. 7) 1932.

Polák discusses the history of the progress of the surgical treatment of pyloric stenosis. The Hildebrand modification of the Rammstedt technic was the operation selected in seventeen cases in a series of nineteen. In the two fatal cases one

patient had congenital infrapapillar stenosis of the duodenum, a rare finding, and peritonitis developed in the other following perforation during a simple Rammstedt operation. These figures and those for another series of two hundred and twenty-six cases with a death rate of 2.7 per cent, in which the same modification was used, compare most favorably with the usual mortality of from 15 to 17 per cent in the Ramstedt series.

STULIK, Chicago.

Miscellaneous

CHILD GUIDANCE AS A MEDICAL FUNCTION. IRA S. WILE, Arch. Pediat. **52**:213 (April) 1935.

This is another of the many articles constantly appearing on this very important question which from its nature does not lend itself to abstracting.

WADDELL JR., University, Va.

THE CHILD HEALTH CLINIC. J. ALBRIGHT JONES and JOHN G. HAND, Arch. Pediat. **52**:259 (April) 1935.

This is a general discussion of the operation of a child health clinic and an analysis of the data on four hundred infants and young children. The authors' summary is as follows:

"1. The child health clinic may have a place in medical centers.

"2. In the operation of such a clinic, an adequate social service department is one of the most important parts to insure satisfactory work.

"3. In this work, cow's milk formula was the one most often used and proved satisfactory. However, advantages of evaporated and dried milk are cited.

"4. Regarding feeding solid food, it is suggested that one of the most important things is never to force a child to eat. Appetite is never created by doctor or mother.

"5. Interesting cases encountered are cited.

"6. A brief discussion of heart murmurs detected at various examinations is given.

"7. A discussion of our results with toxoid and smallpox vaccinations is given."

WADDELL JR., University, Va.

GOLD IN THE TREATMENT OF DISEASE. J. EPSTEIN, Arch. Pediat. **52**:275 (April) 1935.

Epstein discusses the therapeutic uses of gold, particularly as regards its use in the treatment of pulmonary tuberculosis, lupus erythematosus, snake bite, chronic arthritis and whooping cough.

WADDELL JR., University, Va.

INCIPIENT ORTHODONTIC NEEDS. HENRY F. HOFFMAN, J. Am. Dent. A. **22**:680 (April) 1935.

The paper is a discussion of the indications and contraindications for orthodontic treatment and is particularly devoted to conditions which appear during deciduous and mixed dentition. It is designed particularly to assist the general practitioners of dentistry and physicians in forming judgment in cases of malocclusion that come under their observation.

Of particular note is Hoffman's condemnation of a practice now practically obsolete among specialists but still evident in general practice of opening spaces between deciduous teeth when these fail to appear in normal unaided development prior to the appearance of the permanent teeth. A second type of malocclusion, in which there is an inlocking or a lingual position of one or more of the upper teeth, usually requires treatment. Hoffman gives as the conditions which should

be treated: "those types which offer, through the malocclusion of one or more teeth, mechanical interference with development." There are also a brief discussion of the value of space-maintainers when the deciduous teeth are lost prematurely and a discussion of the abnormality produced by the labial frenum. In regard to the latter condition, Hoffman calls attention to the fact that a separation between the upper incisors does not necessarily mean an abnormal frenum. It is his belief that correction should be made by operation only after considerable vertical development of the maxillary and mandibular alveolar processes has taken place. This should not be done, in his opinion, before 8 or 9 years of age, and he thinks that it is wiser to bring the central incisors together by mechanical means before operation.

NOYES, Chicago.

THE USE OF SODIUM FORMALDEHYDE SULPHOXYLATE IN ACUTE MERCURY POISONING. SANFORD M. ROSENTHAL, J. Pharmacol. & Exper. Therap. **54**:34 (May) 1935.

After experimental investigation Rosenthal recommends the following technic: 1. Gastric lavage with a 5 per cent solution of sulfoxylate is carried out, about 200 cc. being left in the stomach. 2. Immediately following this, 10 Gm. of the drug is dissolved in from 100 to 200 cc. of distilled water, and this solution is slowly injected intravenously, from twenty to thirty minutes being permitted for the injection.

In severe cases a second intravenous injection is given, six hours later, of 0.1 Gm. per kilogram of body weight, or a total dose of 5 Gm. If colitis develops later, high colonic irrigations with a 1:1,000 solution of sulfoxylate are given.

The results in twenty-five cases have been excellent, and other clinics have also reported favorable results. The drug reduces the mercuric compounds to the relatively harmless mercurous compounds. The intravenous use is necessary to neutralize the already absorbed mercury and to protect the kidneys.

PILCHER, Cleveland.

USE AND ABUSE OF DRUGS IN THE TREATMENT OF CHILDREN. J. P. PRICE, J. South Carolina M. A. **30**:221 (Nov.) 1934.

The author briefly outlines his observations on practical therapeutic measures for children as follows: (1) Cathartics. A mild laxative is advised, according to the symptoms in the case. The use of mild mercurous chloride and castor oil is distinctly forbidden. (2) Anthelmintics. The first step should be to prove that the child has a parasitic infestation, and then a sufficient amount of vermifuge should be given to relieve the child of the causative parasite. (3) Tonics and blood builders. (*a*) Appetizers are generally not advised. (*b*) General tonics are of distinct value in some cases. (*c*) Blood builders are used in cases of secondary anemia. From 10 to 20 grains (647.9 to 1295.9 mg.) three times a day of iron and ammonium citrate is advised. The value of liver for children is doubtful. (4) Circulatory stimulants. The following drugs are suggested: (1) epinephrine, caffeine, coramine and intravenously injected dextrose. Oxygen, digitalis and camphor are mentioned. (5) Respiratory stimulants. Caffeine, a 25 per cent solution of pyridine betacarbonic acid di-ethylamide and alpha lobeline may be used. (6) Sedatives. Phenobarbital, bromides, chloral hydrate, magnesium sulfate and derivatives of opium are suggested. (7) Antipyretics. Drugs causing the reduction of fever are used too frequently. Hydrotherapy is suggested. Acetophenetidin and acetylsalicylic acid are antipyretics. (8) Specifics. Quinine for malaria, arsenic for syphilis and salicylates for rheumatic fever may be employed. (9) glandular extracts. Insulin and thyroid extract have their place. (10) Serums, antitoxins and vaccines. Smallpox and typhoid vaccines and serums for protection against tetanus, diphtheria and erysipelas and meningococcic and streptococcic infections are given. Pertussis vaccine is suggested.

WESTON JR., Columbia, S. C.

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JOHN D. DONNELLY, M.D., *President, Presiding*

USE OF LIVER EXTRACT IN THE TREATMENT OF INFECTION. DR. R. T. DEVEREUX.

Liver extract, in doses adjusted to the weight of the patient, was given to 23 children, 15 of whom had pneumonia. Four with chronic infection and 4 with acute infection other than pneumonia were given liver. All recovered.

In this series of 23 cases the best improvement clinically and in the blood picture was observed in patients with neutropenia and leukopenia.

In severe infections, when the total leukocyte and the neutrophil counts were sustained at a high level, the intramuscular injection of liver seemed to afford little if any benefit. Otherwise, hospitalization appeared to be shortened by the use of liver during the acute stage of infection.

DISCUSSION

DR. RALPH TYSON: I have had no experience with liver extract in acute infections. Several questions came to mind as I listened to Dr. Devereux's paper, and I should like to ask whether the measures in the two series—those who received liver extract and those who were not given liver extract—were carried out at the same time or whether they were carried out on one series one year and on the other series the following year. It makes a great deal of difference whether one is discussing seasonal cases. For instance, there was a 16 per cent mortality rate for pneumonia at the Temple University Hospital last year, while the year before the mortality rate was 4 per cent. There is no doubt about the fact that the virulence of infections varies from year to year. I think it is true that leukocyte counts vary from day to day without any stimulating drug being given or any attempt being made to stimulate the production of leukocytes. I wish to ask whether all the counts were total counts or whether only a few total counts were made. This is a new idea to me, but it is possible that it will be of help. The series is somewhat too small to enable one to draw definite conclusions.

DR. M. SOLIS-COHEN: I think that Dr. Devereux is on the right track in the treatment of infections. It has been shown that the bactericidal power of the blood is due chiefly to the leukocytes. A great many investigators have shown this. Toplik, of London, has shown that various antibodies, humoral antibodies, have no relation to the clearing power of the blood; the bactericidal power has a distinct relation to this. Fleming, who worked with Wright and who has written a great deal on the subject, has shown that the chief resistance of an individual lies in the phagocytes. In times past nuclein has been used to increase the number of leukocytes, and it may be that liver extract will have an equally good, or a better, effect. This is à subject worthy of further study. Infection is a problem in which one needs a great deal of help.

DR. ERNEST NOONE: May I ask Dr. Devereux what the effect of foreign protein might have been in these cases?

DR. HENRY SANGREE: I should like to ask Dr. Devereux whether any other experimental measures, such as immunologic tests of the perineum and the amniotic fluid and the administration of dextrose and saline solutions, were carried out.

DR. R. T. DEVEREUX: These pneumonias did occur in different years. In both series the distribution of types was about the same—60 per cent bronchopneumonia and 40 per cent lobar pneumonia. Most of the ones such as were treated in the second series had complications of one sort or another. During the period when these occurred 14 patients with pneumonia died in less than eighteen hours. These were excluded from the series.

Although complete hemograms were not recorded in all the cases, all the counts that I spoke of in the paper were total counts.

I have not had any experience with foreign protein. There is nothing to indicate that this is a foreign protein reaction. Physicians in Chicago who gave about five hundred injections could find nothing suggestive of a foreign protein reaction, and analysis of material showed it to be free from protein. As to the use of saline solution in similar series, I have not tried this.

I do not believe that there is anything striking in this method. However, my colleagues and I do think that the period of recovery was much shortened.

STUDIES ON PROPHYLACTIC IMMUNIZATION AGAINST WHOOPING COUGH. DR. HERMAN GOLD.

A pertussis vaccine prepared according to the principles laid down by Sauer, when given to 100 children in the dose and manner prescribed by him, was found to be safe to use. Local reactions occurred in 22 per cent of the cases. Systemic reactions, especially fever and diarrhea, occurred in 20 per cent of the series. Intestinal upset was most common in the younger children (in 53.5 per cent during the first two years of life) and was a rather troublesome complication. The reactions were not severe (except in 1 case). A detoxified vaccine without any impaired antigenic value may be substituted, perhaps with advantage, if this is available, in view of the reactions encountered in this series.

The follow-up work, covering one year, revealed 24 exposures among the immunized children. Fourteen of the children were protected by the vaccine, while 10 contracted the disease (1 case in the latter group is questionable). Of the protected children, 7 gave a history of intimate and prolonged exposure. The rest were transient contacts. Of the children who failed to be protected by the vaccine, 2 contracted pertussis six months after the last injection of vaccine; 2 became ill eight and one-half months later, and 1 became ill seven months later, while 3 contracted whooping cough less than three months after the course of injections was completed.

Prophylactic immunization against pertussis is of definite value, although it does not insure full protection against this disease in all the persons immunized. Prophylactic injections of this vaccine do not affect the course of this disease if the children happen to contract it. The duration of the immunity in those who are protected by the injections of vaccine has not been fully determined. In this series the longest interval between injections and exposure to pertussis without development of the disease was seven months.

DISCUSSION

DR. JOHN REICHEL: I am glad to have this opportunity of discussing Dr. Gold's paper because I may be able to right several matters which may have been misunderstood. Sauer recently reported on the "Known and Unknown of Bacillus Pertussis Vaccine," and this article appears in the November issue of the *American Journal of Public Health*. In the immunization against pertussis one is dealing with a number of variables—the culture, the antigen or vaccine, the dose, the scheme of prophylactic treatment and the immunizing response. Sauer, in particular, is definite as to the culture used. He speaks of his own

vaccine as the "Evanston vaccine" and the vaccines made under agreements as "approved vaccines." The vaccines used by Dr. Gold are not Sauer's approved vaccines. The culture should be recently isolated and one with a smooth colony, or virulent, in contrast to a rough colony, which is not virulent. A smooth colony (virulent culture) is the antigenic type of culture. It should be grown on a medium fortified with 25 per cent human blood. Sauer states clearly the harvested bacteria should not be washed. This is difficult to understand, as there is so much débris included. Our vaccines were washed. As to the scheme of treatment, Sauer's contribution is very definite on this phase, as he uses vaccine of very high counts. It is not clear why the doses on any one day should be given in two injections. I do not think that enough is known about proper spacing to get the best response. Nor does one have the means of telling how long it takes for the immunization response to be effective. One will have to get such information to determine the extent to which the child is protected or susceptible to exposure. About 10 per cent of those who received the approved commercial vaccines contracted pertussis when they were subsequently exposed, according to Sauer.

Dr. A. M. Dannenburg: Those who have children under their care are anxious to have them protected against pertussis. For many years I used these mixed cultures or vaccines with little success. After the publication of Sauer's article I again used vaccine in the children under my care. Of the first 27 children given an injection, 8, or 29 per cent, contracted pertussis four months after they were inoculated. This was such a high percentage that I took the matter up with the commercial concern from which I purchased the vaccine. The president and chief bacteriologist admitted that for the first few months the vaccine was nothing more than a stock culture. Subsequent to this I used the approved vaccine, and to date I have inoculated 125 children, 102 of whom are beyond the four months' period. Three were intimately exposed and did not contract the disease. To date no mother has notified me that her child has come down with pertussis. I have not called or written the parents as yet, but I plan to do so. It is extremely interesting that 29 per cent of 27 children who received an indifferent, or stock, culture contracted pertussis, whereas 102 children who received the approved vaccine did not, and of these 102, 3 were definitely exposed.

Dr. Julian Lyon: I should like to ask whether it might be possible that a vaccine prepared by a different laboratory in a different part of the country might not have the characteristic of producing intestinal disturbances, as was shown in this series. I do not have nearly as many patients as Dr. Dannenburg, but I do not remember that any of my patients reported that they had intestinal disturbances. I used vaccine from a different laboratory. I should also like to ask whether the incubation period is prolonged in these children who have been immunized, if they do contract whooping cough.

Dr. Theodore Melnick: Like the previous speakers, I am using the approved vaccine. At first I obtained some gastro-intestinal reactions; I then resorted to the procedure which I had advocated a few years ago with scarlet fever vaccine. This was before the use of the approved vaccine. My reactions have been nil with the approved vaccine, whether owing to the use of the approved vaccine or to procedures that mothers follow before giving the vaccine. My latest patients have been entirely free from any gastro-intestinal disturbances; there is merely a slight local reaction—a small degree of local redness or tenderness. None of the patients so far has come down with the disease, though 10 of them have been in absolute contact with it.

Dr. Pascal Lucchesi: At the Philadelphia Hospital for Contagious Diseases we are treating with vaccine as a routine all patients admitted with some complications. We are using the approved vaccine and not the same vaccine that Dr. Gold used. We have failed to find the high rate of diarrhea that he noted. In those children exposed in the ward—patients with secondary cases—we again failed to note the high incidence of diarrhea. Another point which seems to be of interest is the high number of those who contracted the disease after they had

been given an injection. I think that of 100 children who are definitely exposed, about 25 per cent should escape the disease without being given vaccine, in spite of the fact that they are exposed. It also seems to me that a possible factor may be the difference in the vaccines; apparently the approved vaccines are not giving the same results.

DR. JOHN P. SCOTT: I have a series about as large as Dr. Dannenburg's and have not observed any gastro-intestinal reactions of the type mentioned. It seems to me that this immunization may rebound in one of two ways: First, an incomplete immunity may produce subclinical and unrecognizable conditions, and the subjects may act as carriers and infect the untreated population; second, the immunity may be of short duration and allow the occurrence of numerous cases in high schools and business establishments, with costly loss of time. As a public health measure, much depends on the universality of inoculation, as is the case in diphtheria. Its value is great in the younger children, to whom pertussis is a real danger.

DR. EDWARD THORPE: I should like to ask Dr. Reichel some questions. My recollection is that the typical organism of pertussis isolated from a fresh culture is a rather long, slender, irregular type of rod and that after growing in successive cultures on artificial mediums it changes its form and becomes a shorter and thicker rod, giving a smooth type of colony. It does better, however, on the blood plate which Dr. Reichel said that he and his associates are using, the Bordet medium plus 25 per cent of human blood. I should like to ask him whether he can recall the morphologic features of their organism. In regard to a second question: He said that they are washing the colonies when they prepare the emulsions. It seems to me that in the case of all those cultures grown on blood plates washing the organism may remove some of the products of red cell decomposition, and possibly some of the ill effects may have come from that source. A common method in use of removing these colonies where blood has been used is scraping with light glass scrapers. There is also a chance of removing some of the débris of the culture mediums. I do not know whether it is technically possible to remove the débris also, but certainly one would not get any of the products of hemolysis. I should like to ask Dr. Reichel whether he can enlighten the members on that point.

DR. J. STOKES: I should like to ask Dr. Gold whether he believes that his vaccine compares with Sauer's vaccine and whether it has any effect on antigen. Also, I wonder whether those vaccines taken by washing according to the Zinsser method might remove something from the point of view of the antigen.

DR. HERMAN GOLD: In our study, my associates and I were struck by the instances of failure in the series. We thought that the diarrhea was perhaps due to the high bacterial content of the vaccine. We checked the so-called approved Sauer vaccine's bacterial content as compared with that of the vaccine that we were using and found perhaps what may be a clue—that the so-called approved vaccine dispensed on the market does not contain the high bacterial count that it is supposed to contain. The count does not come up to 40 per cent of the alleged count. In using the commercial approved vaccines, instead of injecting 10,000,000,000 organisms, one is really injecting only 4,000,000,000, which may be one reason for so much diarrhea in our series. Dr. Sauer now acknowledges quite a few failures in his series. Whereas he originally reported 100 per cent success, he now reports 8 or 10 cases of failure, and perhaps he may state a higher incidence of failure later on.

DR. JOHN REICHEL: I am afraid that the speakers have drifted far in this discussion, but I wish to say that the "unapproved vaccine" was made from Sauer's cultures, as received from him for that purpose. A culture which is cultivated as a stock culture does change in morphologic features. A culture when first isolated may have intangible qualities.

In regard to Dr. Stokes' question "What is in the vaccine?" the answer is: some specific protein carbohydrate or polysaccharides. The free polysaccharides should be removed, as they lower the resistance. I think that the general trend of

the present knowledge confirms the opinion that the immunizing power is the specific protein in the antigen. It is possible to make an antigen by taking a washed suspension of the organism and grinding it, as Kreuger, of California, has done.

ENURESIS: A STATISTICAL AND ANALYTIC STUDY. DR. SIDNEY WEISS.

One hundred and eighty-seven children and young adults with enuresis were studied to determine the etiologic factors and their significance in this disorder. The case records of 16 patients were then analyzed. Statistically, the intelligence quotient and the age, as well as the economic status, were found to be important. Poor habit training, parental rejection, step-parent-child relationships, jealousy, fears and other factors of a psychic nature were found to be causes of enuresis.

DISCUSSION

DR. GERALD PEARSON: I was glad to have the opportunity to hear Dr. Weiss' paper because, as a psychiatrist, I must confess that there is no problem quite so baffling as that of enuresis. It is so baffling that when Dr. Weiss talked with me about his study of enuresis it seemed that it might be a good plan to try to determine what factors may or may not have an etiologic significance, and I think that he will agree with me that his study is simply an effort to determine this. His study brings out three or four important points: First, there are two types of enuresis—that which occurs after a dry period and that present from birth. In these two types the causation and the whole problem are different. In the type which is present from birth there are probably several subgroups with different causes. I should like to make clear that what he considers to be enuresis is not the type of incontinence which has a well known and distinct physiologic basis. Thus the type of enuresis that is present from birth is not a physiologic problem, because there is no physiologic reason why a child should not wet himself in the daytime if he wishes to. Among certain primitive peoples a beautiful baby is one covered with feces. In such a community enuresis would not be a problem, so that the type of enuresis that is present from birth seems likely to be a question of the training of the child. I believe that among the cases of enuresis that is present from birth there are a number of instances in which the child has received no training or inadequate training, and in my experience a certain number of parents do little about the training of their children. In this connection Dr. Weiss' finding that enuresis is more common in children of parents of low economic status and a low intelligence level is important. Probably the mothers, hard pressed by financial conditions, with poor accommodations and low intelligence, do not bother to train their children in toilet habits even up to the age of 8 or 9 years. Dr. Weiss' statement that there is a group of children with this type of enuresis who have a definite type of personality, being passive, nonaggressive and babyish, is also important. In these cases the problem seems to be the suppression of aggressiveness, although it is not very clear, even to the psychiatrist, why, if a little girl gets very angry with her mother, she wets herself at night rather than expressing her anger directly. This is a problem which needs further study.

In a third group of cases of enuresis that is present from birth, the enuresis probably represents certain fantasies, although Dr. Weiss has not gone into this aspect. Another point which Dr. Weiss did not bring out but which is also important is that enuresis may run in families; that is, there is a constitutional factor in certain cases. Enuresis is not a problem which should be passed over lightly with the simple prescription of an alkali or atropine, but the case needs intensive study from the sociologic and psychologic aspects. When this is done in more cases it will be possible to determine the subgroups of enuresis that are present from birth and what etiologic factor is important. The enuresis which occurs after a dry period has been more intensively studied; its etiology is purely psychologic, and the treatment also must be psychologic.

CHICAGO PEDIATRIC SOCIETY

Regular Meeting, Jan. 21, 1936

ABRAHAM LEVINSON, M.D., *President, Presiding*

RATIONAL TREATMENT FOR ABNORMALLY ATTACHED FRAENUM LABII. DR. A. B. SCHWARTZ.

The fraenulum labii superioris is a band of tissue that connects the back of each lip to the front of the gum. The upper frenulum is the better developed and may be brought into view by everting the upper lip. The idea has prevailed that the attachment of the frenulum between the incisors produced abnormal spacing between these teeth, with resulting malocclusion. Plastic operations for the removal of the frenulum were devised. Some physicians urged early operation to obviate the need for orthodontic treatment, while others advised operative procedures only after orthodontic appliances had proved unsuccessful.

By abnormal frenum is inferred a thickening of the band, and its attachment to the margin of the gum line, or, in some cases, curving of the frenum under the gum and attachment to the lingual aspect of the mucous membrane of the gum. Besides actual thickening of the fibers, in some cases the frenum shows a knobby overgrowth just at its insertion on the margin of the gum.

Advice received by several patients to have an excision of the labial frenum prompted me to note the frequency of an abnormal frenum in children seen in office practice. After inspection of numerous office patients it was evident that so-called abnormal frenum is exceedingly common in infancy.

With the assistance of Dr. Thomas Abbott, head of the dental department of the Milwaukee Children's Hospital, my associates and I inspected the mouths of the children of two Milwaukee schools. For this purpose we selected grades with pupils of varying ages. Among 116 children varying in age from 3 to 6 years, inclusive, there were 26 with sufficient thickening of the frenum and low attachment so that the condition could be designated as abnormal. There were 15 children with separation of the upper central incisors. Eleven children had missing incisors, so that it was not possible to make any statement about separation of the incisors. Among 128 children from the third to the seventh grade, inclusive, the ages of the pupils ranging from 9 to 13 years, there were only 8 with an abnormal frenum, 4 showing separation of incisors. In the 13 year group alone there were 62 children, 3 of whom had an abnormal frenum, 1 showing separation of incisors.

From the results of examination of this group the conclusion seemed warranted that as the child grows older the attachment of the frenum is found farther away from the gingival crest, and the presence of separation of incisors is less frequent.

This is in accord with the opinion of conservative dental authorities, whose views may be summarized as follows:

1. There is insufficient evidence that the abnormal frenum has anything to do with separation of the upper incisors or with malocclusion. Often such separation may be seen between other teeth and is of familial character.

2. The central incisors, when separated, are more easily moved together by appliances before scar tissue is formed, and, conversely, are moved together with more difficulty after scar tissue is formed.

3. With the eruption of the full complement of the permanent teeth the condition will correct itself.

The correction of abnormal spacing between the incisors by mechanical appliances rather than by excision not only seems more logical but is in accord with the embryologic nature of this abnormality. Bolk, in a careful study of the embryology of the oral structures, demonstrated that in the development of the vestibulum oris the frenulum is pushed up as an intermediate bridge. He pointed

out that at one stage of development the upper lip is in reality a right and a left lip, a condition actually present in some of the lower primates. Bolk stated that in cases of widening of the frenulum the condition should be regarded not as a hypertrophy but as a developmental defect in which the fusion of the two sides of the vestibulum oris has not been completed. In those instances the strip of tissue for the development of the teeth is equally incomplete. From this it would seem to follow that the excision of the frenum alone is not the indicated procedure, but such orthodontic measures as will bring the central incisors together are necessary. Excision of the frenum, with the resultant scar tissue, would seem as definitely contraindicated on embryologic grounds as it seems to be from our clinical observation.

CLEFT PALATE AND CLEFT LIP: THE SEQUENCE OF OPERATIONS FOR SURGICAL CORRECTION. DR. WILLIAM H. G. LOGAN (by invitation).

A discussion of the surgery of cleft palate and cleft lip before an organization composed entirely of pediatricians, although a bit unusual, is nevertheless appropriate, since each pediatrician is called on from time to time to advise the parents of children born with congenital cleft of these structures. The query is often raised as to the time that surgical intervention should be instituted in order to secure the best results in accordance with the prevailing opinions of the leaders in surgery in this particular field. To avoid a lengthy discussion and to confine myself to the time specified, only a declaration of the principles involved will be made; this will be followed by a brief substantiating discussion.

The three questions most frequently put to the pediatrician with regard to this congenital deformity are as follows: 1. At what age, by preference, should surgical intervention be instituted? 2. What part of the deformity should be corrected first? 3. What should be the sequence and time of the necessary subsequent operations?

An infant born with cleft lip (complete or incomplete, single or double) but having a normal maxillary ridge which may or may not be complicated with a cleft of any degree of the hard and soft palate should not have the deformity of the lip corrected, as a general rule, until it has ceased to nurse (provided that it is able to nurse) unless the physician in charge is positive that the mother's milk can be maintained throughout the period of postoperative treatment. In cases in which the maxillary ridge is normal and the infant is not nursing, the operation on the lip need be postponed only until a proved formula has been found, a satisfactory physical condition has been established and an appropriate daily gain in weight has been maintained. After the closure of cleft of the lip in this type of deformity, one is confronted with the problem of determining the appropriate time for the operation necessary to close the cleft that extends to any degree through the soft palate and likewise that extends to any degree through the hard palate up to the normal alveolar ridge. In practically all instances closure of this cleft, regardless of its extent, should be accomplished in one operation at some period between 1 year of age and the time that the child begins to speak; as a rule I prefer to operate on these patients some time between the fourteenth and twentieth month.

The next question relates to the time and sequence of operations on a child born with a cleft of the lip complicated with complete cleft of the hard and the soft palate and the maxillary ridge. In the surgical correction of such a congenital deformity I believe that the best results will be more uniformly secured if the first operation deals with the closing of the cleft through the maxillary ridge and the anterior half of the hard palate by means of a turnover flap either from the vomer or from the overlying soft tissues from the long fragment to the short and a reduction in the width of the cleft throughout the hard palate when indicated. Three or four weeks later the operation for closure of the cleft of the lip is performed.

The operation for the closure of the bony cleft through the maxillary ridge and for reduction of the width of the cleft through the hard palate should never be performed before the infant is 2 months of age, and then not until the child has been placed on a proved formula, has regained its weight at birth and is making satisfactory daily physical progress. The feeding should be carried on under the direction of a specialist. When the operator is permitted to set the time arbitrarily for the first operation for these complete clefts of the upper jaw, a date between the second and the fourth month is selected, without regard to whether the condition is complicated with protruding premaxillary bones. From three to four weeks after the operation for closure of the cleft through the divided anterior maxillary ridge and at least the anterior half to one third of the hard palate, the cleft in the lip is closed. Some time between the twelfth and the twentieth month an effort is made to close the remaining cleft in the hard and the soft palate in a single operation.

The time and sequence of surgical corrections for congenital cleft of the lip and palate may be briefly summarized as follows:

Cleft of the Lip, All the Other Structures Being Normal.—If the child is nursing the lip should not be operated on until the infant has been weaned, unless the physician in charge is positive that the mother's milk can be maintained during the postoperative period. If the child is not breast fed, surgical correction of the lip may be made any time after the weight at birth has been regained and a satisfactory physical condition has been established.

Cleft of the Hard or the Soft Palate of Any Degree, with a Normal Maxillary Ridge and a Normal Lip.—The surgical correction of such a cleft should be undertaken some time between the twelfth and the twentieth month, with preference for a period between the fourteenth and the twentieth month, this to be accomplished in a single operation.

Complete Cleft of the Upper Jaw and Cleft of the Lip of Any Degree.—When such a deformity is present the first operation should be performed between the second and the fourth month. This should include closure of the maxillary ridge and the anterior one third to one half of the hard palate by means of an everted flap, this to be accomplished by the direct application of force through the employment of silver wires and lead plates. Then three to four weeks thereafter, if the physical condition of the patient warrants, the cleft in the lip is closed. The remaining cleft in the hard and the soft palate should be closed some time between the twelfth and the twentieth month, with preference for a period between the fourteenth and the twentieth month.

By not operating on the lip, when there is a complete division of the upper jaw, until the approximation of the maxillary ridge, and, when indicated, that of the separated bones in their anterior one third, has been accomplished by means of an everted flap from the long to the short fragment, one is able to construct a more nearly normal floor of the nasal fossa on the afflicted side.

Since the nose of a patient with a single cleft of the lip and a complete cleft of the palate is not, as a rule, in the median line and can more positively be brought into its normal position and retained there by bending the long side of the bone toward the fissure, at the same time pressing it backward to overcome the usual labial prominence that practically always obtains, one can, by operating during the period when the bones will bend, carry the center of the nose to the median line and fix it there.

A more harmonious outline of the maxillary ridge and bony facial support can be secured when the separated bones forming the upper jaw are placed in their proper position in the first weeks of life, but great caution must be exercised not to carry the protruding bone on the long side too far inward with the single thought of securing harmony of outline of the maxillary ridge, for to do so often results in bringing the upper anterior teeth inside the lower teeth and giving to the upper lip a depressed appearance that can be avoided when the proper technic is employed in this operation.

By operating in early infancy development of the jaw and of the face will be more symmetrical than would be the case if these bones, soft tissues and muscles were not brought into full functional activity until some years after birth.

It has justly been contended by advocates of early operation for correction of cleft palate that if the child is to be expected to appear and speak normally the abnormality should be overcome before the infant begins to speak. If one forms for the child a normal functioning palate and lip before it begins to articulate words, one can with greater justification expect it to speak as other children do who are born with these parts normal.

An earnest plea is made that the preoperative and postoperative medical treatment of these infants be supervised by a pediatrician. If the surgeon makes it a rule never to operate on such patients until they are pronounced physically fit by a specialist, his percentage of mortalities will be exceedingly low. My records show a more serious reaction following operations on the lip than following those on the palate.

The procedure of bringing the separated bones of a complete cleft of the maxillary ridge and hard palate at its anterior one third to one half into apposition and, when necessary, reducing the distance between the borders of the cleft straight in from the tuberosities to a degree that the condition demands and there retaining them until union occurs calls for the application of force at given points and on fixed lines and retention with materials that will withstand the strain necessary to hold these separated bones in contact without undue irritation for three or four weeks. The principle for closure of complete cleft of the upper jaw as advocated by Dr. Brophy and as modified by me (described in the Transactions of the English International Dental Congress, Paris, France, 1931) is recommended because it gives a higher percentage of success than any other technic with which I am acquainted. For the past few years I have been led to believe that lead plates, when utilized for the closure of the cleft in the hard palate, should not be retained for a maximum of from six to eight weeks, as practiced by some operators, as they may interfere with osseous development of the divided jaw.

Although only a single technic has been suggested, I do not contend that other methods will not frequently produce satisfactory results, provided the following rules are observed: 1. The method followed should preclude the making of incisions that will destroy or reduce the function of the important muscles of the palate. 2. The operative procedure should obviate, so far as possible, any unnecessary shortening of the palate. 3. The method adopted should result in a reduction of the distance between the borders of the cleft of the bone directly in from the tuberosities, when indicated. 4. When the cleft is complete the surgical correction by means of an everted flap should establish the maxillary ridge and the floor of the nasal fossa, at least in its anterior part. 5. The correction of the cleft in the lip should permit it to function relatively normally, and it should be symmetrical in appearance and thickness with the tissues beneath the nostril on the normal side.

One should be ever mindful that not only the palate, but also the lip, must be given sufficient fulness to meet the lower lip so that a patient will be able to say such words as "boy"; "Peter"; "bat"; "papa"; "lip," and "back." Unless the maxillary ridge is properly formed so that the teeth may erupt relatively normally or may thereafter be normally supplied, these patients will be unable to enunciate such words as "vain"; "vanity"; "front"; "fast," and "farthest."

If the condition is complicated with protruding premaxillary bones, these should never be excised but should be retained and placed in position by a submucous bone incision ¼ inch (0.6 cm.) above the junction of the vomer back to the point where the usual resection of bone is made to permit the premaxillary bone to be drawn downward and placed in contact with the lateral portions of the maxilla. The old method results in extreme flattening of the end of the nose. I have found it possible to correct materially this objectionable feature by making the incision in the line indicated, which allows for carrying the premaxillary bone down-

ward and backward and stabilizing it in a relatively normal position, in this manner reducing the usual amount of flattening that so often occurs at the end of the nose in these cases.

I wish to voice approval of the idea expressed by Rosenthal that "the ideal purpose of a cleft palate operation is the complete reconstruction of a divided cleft not only anatomically but physiologically." I also desire to admit, as do many other students of this subject, that the physiologic results of the operations performed by my associates and me, so far as they are related to speech have not been all that they should be, and that has been the activating motive in my study. All those interested in this field who have not already done so should acquaint themselves with the management of congenital cleft of the palate by Victor Veau, of Paris, whose work compares favorably with the leaders not only of the present time but of all times. Furthermore, we extend appreciation to that small group of surgeons—Franz Ernst, of Berlin; Wolfgang Rosenthal, of Leipzig; George M. Dorrance, of Philadelphia, and Alexander Limberg, of Leningrad—who have made such painstaking efforts to gain the end-result of better speech through maintaining the repaired vault in the palate at a higher level or through reducing the distance that usually obtains between the repaired soft palate and the posterior wall of the pharynx.

Our prime interest through the years has been to find the best way to correct the congenital deformity of cleft lip and palate so that persons so afflicted may take their places in society without discomfort and embarrassment. I think that we are also agreed in the principle that the construction of a normal-appearing lip, with a normal facial expression, is but a single important item. This should be accompanied by satisfactory speech; otherwise, failure in part will be the result, whereas success might have crowned one's efforts if one had been a more conscientious student of the entire problem.

PRIMARY AND MAJOR INDICATIONS FOR ORTHODONTIC TREATMENT. DR. THOMAS L. GRISAMORE (by invitation).

In presenting the subject assigned I thought it advisable to show slides illustrating various conditions requiring orthodontic treatment. The patients selected come within the age limit of those seen by the pediatrician.

It should be remembered as an underlying principle that if a growing child is to have eventually the dental arches and physiognomy that nature intended that he should have, the forces that enter into the development of these parts must function normally. Regardless of the age of the child, if there are signs of present or potential faulty development of those parts due to abnormal function of the developmental forces, treatment should be instituted. If this is not deemed advisable at the time, casts should be made and the patient kept under observation.

The deformity of these parts depends largely on the length of time that the forces of growth and development are permitted to function abnormally. And the ultimate result of orthodontic treatment depends almost entirely, so far as the dentures are concerned, on the equal balancing of force. Function determines form. Well developed facial muscles are more beautiful than those not so well developed, and the correction of conditions by myofunctional treatment added to the mechanical therapy will in most cases produce better results and materially lessen the liability to relapse.

Orthodontic treatment should be instituted as early as possible after it has been decided that the anomaly will not be corrected by the natural forces. Better results usually will be obtained if the treatment is started at an early age, but the term of treatment is liable to be of longer duration in such instances. In most cases treatment is not completed until after all the permanent teeth, with the exception of the third molars, are in position.

There can be no arbitrary rule for the time of starting treatment. This must be determined by visualizing future developments in each case under the existing conditions. The state of development and not the age of the patient should be

the guide for orthodontic intervention. The age of the patient has little significance, as children of the same age may be at various stages of development, depending on the general health, the hereditary influence and other factors.

It is not uncommon for a mother to bring in her child for correction of an oral defect long past the time when orthodontic treatment should have been given, stating that she had been advised to postpone treatment until all the permanent teeth were in position or that the anomaly, if left alone, would correct itself. Such advice has no doubt been the reason why many persons go through life with oral and facial conditions far different from those intended by nature. Children suffering from abnormal respiration due to adenoids and enlarged tonsils who are permitted to develop until maturity or well past the age of puberty before treatment is instituted can never have the anomalies completely corrected. After this time the only part of the physiognomy amenable to treatment is that influenced by the position of the teeth and their supporting structures.

I realize that the members of the Chicago Pediatric Society are not especially interested in orthodontics, but they are interested in children and their welfare, and I sincerely hope that the slides that I shall show will be of some interest and value to those present.

These slides show the influence that the various forces have on the development of the arches and on the physiognomy. The eruptive forces exert an influence on development from before birth until the entire permanent dentures are in position, which is early manhood or womanhood. Nature intends that the deciduous dentures be kept in position until the permanent dentures are ready to take their place. A number of these slides show the evil effects on the permanent denture and that part of the face sustained by the dentures and their supporting bony structures from the too early loss of the deciduous teeth.

Similar conditions, before and after treatment, are shown, illustrating the advantages of instituting treatment at an early age in cases in which the developmental forces are functioning abnormally. By early treatment many conditions which, if left alone, would develop into a decided deformity may be corrected with little trouble. Other types of anomalies are shown to demonstrate those that would probably be corrected by the natural forces, and some anomalies that appear similar but that no doubt would require mechanical intervention are shown.

These slides were selected with the object of stressing the necessity for keeping under observation during the developmental period all the forces that enter into the shaping of the dental arches and facial outline. Bones no doubt grow to their inherited size but their form is influenced by the forces exerted.

DISCUSSION

ON PAPERS OF DRS. SCHWARTZ, LOGAN AND GRISAMORE

DR. HENRY E. IRISH: I have been much interested in this presentation, not only from the surgical aspect but from the standpoint of one who has been in the position to which Dr. Logan referred as taking the responsibility for the pediatric preparation and after-care. For many years I have had charge of the preparation and the after-care of children that come into Cook County Hospital with dental anomalies. I learned some facts, and many remain to be learned. It is with the hope of receiving some suggestions concerning the facts yet to be learned that I speak.

My associates and I have learned that the patients who are operated on for cleft palate have strange sequelae. We seldom observe a case in which a temperature of from 103 to 104 F. does not develop within a few hours. The high temperature is accompanied by a profuse nasal exudate, and this is prolonged, as a rule, for about a week, in spite of anything that we have been able to do. I have seen 3 successive patients die of pneumonia in three successive weeks. Some measures that we have taken seemed to help. We have given these children isolation and complete nursing care of the kind given patients with contagious diseases. That means that they are either in separate rooms or in company with 2 other children who are being treated for the same condition. In this instance they are isolated from each other by screens and attended by nurses who wear masks

at all times. Unfortunately, we are not able to mask physicians and therefore do not have full isolation from the standpoint of nursing care for patients with contagious diseases. With these precautions we have seen considerable improvement, and I believe that if full aseptic technic were instituted for at least a week before and a week after operation the results would be even better.

In my private work it has been my misfortune to have operated on 3 patients for cleft palate, 2 of whom I isolated for a week before operation in a private hospital but who did not have a private nurse. We did not exclude the family. One of the children operated on had a high temperature immediately and two weeks of severe and stormy convalescence. The result was about 50 per cent union. Another patient was brought in during the evening, was seen by my resident physician and was operated on the following day at about 10 a. m. I did not see the child. The patient had no fever until 2 p. m., when the temperature was 103 F.; by 6 p. m. it was 106 F. The next day at 3 a. m. the temperature was 107 F., and the child died. Postmortem examination showed what the pathologist regarded as pneumonia of all of both lungs excepting the upper lobe of the left lung. To me the condition looked like acute congestion in both lungs, and it suggested that there may have been some relationship between traumatizing the tissues of the nose and the condition in the lung. It is known, of course, that there is a relationship between irritation in the nose due to hay fever and disease of the lung, and it is barely possible that in this case some infection was present which was not shown by the temperature or on examination.

The third child was prepared by being in the ward for fifty-seven days. No temperature higher than 100 F. was recorded for eighteen days previous to the operation. The patient was in a private room for four days previous to the operation, yet she had a temperature of 104.8 F. ten hours after the operation and a temperature of 103.4 F. six hours after the operation. It was nine days before the temperature became normal (under 100 F.). There was a profuse exudation, which was serosanguineous, then purulent. The result was maceration of tissue, sloughing of all the sutures and slight union.

In the future I shall insist on asepsis for at least a week before operation in such cases, much more rigid than I have practiced heretofore. I should like to know whether any one here has used antiseptics and has made any attempt to remove any organisms from the mouth and nose before operation. When it is remembered that these structures are open to food that is put in the mouth and are open above to anything that may be inhaled in the air it is difficult to assure any degree of asepsis following operation. I should still expect, as a result of traumatizing the nasal mucosa, profuse exudation, with maceration of tissue and fever either from trauma or from absorption.

Dr. Charles Schott: The time of the year, in my experience also, has had no influence on the results following operation in cases of harelip and cleft palate. In the cases that have come under my observation for a number of years the results have been as good in fall and winter as in spring and summer.

It is thought that the proper preparation of these infants and children has had much to do with the satisfactory outcome. It has been the rule, of course, to have them in a proper state of nutrition. They must be gaining weight properly and show in their general state of well-being that they are on the up-grade. Then, for a week or ten days before operation, alkalinization has been instituted. Immediately on completion of the operation a 5 or 6 per cent solution of dextrose has been given to them by hypodermoclysis while they are still on the operating table. This injection has been given into the breasts and into the axilla.

With the new operation of the closure of the bony arch and the hard palate in one stage, the reaction has not been great. A rise in temperature for perhaps a day or two to 102 F. may be expected. The same is true with the repair of the lip. Usually after that time no trouble should be expected. With the closure of the soft palate it is not unusual to have more trouble; this should be expected when one recalls the interference with Rosenmüller's fossa and the eustachian tube,

and otitis not infrequently develops within the first week, which will give the temperature an added boost, not because of a new infection but because of the bacterial flora present in the mouth and throat.

Let me again emphasize that the time of the year has nothing to do with results in the management of this type of condition postoperatively.

DR. ASA J. LAGROW: I have enjoyed these three papers very much. In response to the president's invitation, as a member of the dental profession, I should like to discuss briefly the paper by Dr. Schwartz. The subject was well presented by the essayist. Management of the abnormal frenum of the lip often presents a problem to the orthodontist.

The function of the labial frenum is to hold the lip in normal position during the act of laughing or smiling. The normal labial frenum is reflected from the lip on both sides of the median line to the mucous membrane covering the alveolar bone and continues vertically toward the gingival border between the central incisors and then lingually to a point near the dental papillae.

Histologic examination reveals that the labial frenum is made up entirely of fibrous connective tissue fibers. The point brought out by the essayist that the space between the central incisors becomes less as the child grows older is a good one, and this does take place in those cases in which the frenum is normally attached. In cases in which one finds the deciduous central incisors widely separated owing to a heavy labial frenum, I agree with the essayist that the patients should not be treated. As the roots of the deciduous teeth are resorbed the alveolar bone investing those roots is also resorbed. At this period of development, which varies from the age of 5½ to 7 years, the maxilla increases in length and depth, and as the permanent teeth erupt new alveolar bone grows down to invest those teeth. After this rapid change has taken place it is found, in normal cases, that the attachment of the frenum has not grown down with the permanent teeth but has remained where it was in the deciduous teeth, thus producing a normal labial frenum with a normal contact of the permanent central incisors.

However, in some cases an abnormally heavy frenum does grow down with the eruption of the permanent teeth and causes a permanent diastema if the condition is not corrected. In some of these cases the axial alinement of the permanent central incisors is disturbed sufficiently to cause a disfigurement. The tough fibrous tissue of the frenum prevents the fibers of the peridontal membrane, which pass from tooth to tooth gingivally to the alveolar crest, from drawing these teeth to a normal contact. There are two different methods for managing these conditions. One is to bring the teeth together with a mechanical appliance, thus compressing the fibrous tissue, with the hope that it will be resorbed. The other method is to resect the fibrous tissue causing the separation, leaving a normal frenum, and then bring the teeth together mechanically. In those cases in which the fibrous tissue is especially heavy I prefer the latter method. I have seen many of these patients several years after Dr. Thomas L. Gilmer resected the correct amount of tissue, and I have never found any scar tissue.

DR. LOUIS MINSK: I should like to know how many of these deformities need this orthodontic work. I think that psychologists now minimize thumb-sucking as being of any consequence in these cases.

DR. CRAIG BUTLER: I have been observing in new-born infants an anomaly that occurs rather frequently; that is a deep notch in the middle of the upper gum where the frenum attaches. In about 3,000 babies seen at the Cook County Hospital I have found that this notch occurs three times as frequently in the Negro infant as in the white infant. I have one child under observation in whom the notch was very broad; it seems to be gradually filling in, and I shall be interested to see how much separation remains when the permanent incisors erupt. I think that as the child grows older it will draw together.

DR. E. T. MCENERY: Does Dr. Logan select any special time of the year for these aspirations? Acute infection of the upper respiratory tract in children with cleft palate is very prevalent during the fall and winter months, and it is

difficult to prepare these children for operation at this time of the year. In fact, it is not uncommon to reject 5 or 6 children every day that the clinic is open who are brought in for operation, because of slight infection of the upper respiratory tract. If these infections are present it is much more difficult to get a complete closure after operation. I have felt that because of the poor results during the winter months it is better to discontinue operating during this season.

Dr. I. M. Levin: Will Dr. Logan tell us whether or not Brophy's teaching that the "upper jaw bones are not, as a rule, defective in structure nor incomplete in development. There is only abnormal elevation of the palate and failure of union" is still accepted?

As was emphasized by Dr. Irish, adequate preparation of the patient is of paramount importance. A specialized technic of feeding must have been worked out to insure the nutritional requirements of the infant. Preoperative starvation and dehydration must not exist. In no field of surgery is it more essential to handle tissues with gentleness and sureness. In the best hands the incidence of secondary infection and failure is only too frequent.

The errors in the field of orthodontia may in a large part be laid at the doors of the medical and dental professions. The teaching in the medical schools is entirely inadequate. The student is unfamiliar with normal dentition and occlusion. The dentist errs in advising a policy of delay in the care of problems involving deciduous teeth.

The physician should familiarize himself with the abnormal habits of the tongue, cheeks and lips, which produce shifting of the teeth, malocclusion of the jaws and regrettable facial defects and asymmetry.

Dr. Mandel L. Spivek: I cannot agree with the preceding speakers. I do not think that a period of hospitalization is safe if used as a period for building these patients up. Unfortunately, these children have to have these operations performed in a children's hospital and, as is well known to pediatricians, such a place is probably the worst environment that they can possibly be in. To have a child come in for an operation for cleft palate and remain in the hospital for a week before surgical intervention seems wrong to me. They should be hospitalized for possibly two days preceding operation, and then if they show no departure from the normal the operation may be performed. I think that the physician should be the person to decide whether surgical intervention is safe at that period. It is almost impossible to prevent infections of the upper respiratory tract if they are present in the hospital and its personnel. I do not believe that any technic can prevent the spread in this case. Each day of hospitalization adds to the danger of infection. At the Children's Memorial Hospital these children are kept in a room absolutely to themselves. No other patients are admitted to these rooms, yet in spite of these precautions, and in spite of a personnel that is educated to the dangers of their own slight colds and regardless of a good nursing technic, infections of the respiratory tract spread. The danger of this initial infection of the upper respiratory tract is well known.

Dr. William H. G. Logan: Before replying to the questions that have been raised by the various discussers, I ask that I be permitted to refer briefly to the splendid presentation made by Dr. Schwartz on the subject of the abnormally attached frenum of the labium superius, in which he pointed out that there is serious danger that scar tissue may form between the superior incisors when the frenum is surgically removed. In my opinion, surgical intervention is indicated in only a small percentage of cases; furthermore, when there is a demand for excision of the frenum of the labium superius I have never felt called on to carry the incision between the teeth but have always confined the line of incision to the labial surface of the upper jaw. In this way the normal gingival tissue between the teeth and the labial surface never is injured.

The question has been raised by a number of the speakers as to the propriety of performing operations for cleft palate and cleft lip during the winter months. The contention has been that in children with these conditions involvements of

the upper respiratory tract frequently develop after admission to the hospital that contraindicate operation, and for that reason the children are sent home rather than retained in the hospital until a satisfactory physical condition has been established for operation. With regard to the question as to operating in the winter versus the summer, I believe that all surgeons have a preference for avoiding surgical operations during the severe winter months. However, if there is indication that the appropriate time has arrived for the operation for cleft lip or cleft palate, it has been my custom to have the patient brought to the hospital and placed in charge of a pediatrician. When the child is pronounced physically fit by the pediatrician we proceed with the operation, and our results have been such as to justify continuation of operating either during the winter or summer, provided we abide by the instructions of the specialist. However, it should be added that when these operations are performed in the winter months it is sometimes necessary to keep the patients in the hospital for a few extra days before the pediatrician pronounces their condition safe for operation. The point that I wish to make clear is that if as good a physical condition of the patient can be established in the winter as in the summer we do not hesitate to proceed with the operation, nor do we anticipate a higher percentage of mortalities. However, by preference the extreme of winter should not be looked on with favor for this or any other surgical operation.

With regard to irrigating the mouth and the field of operation with sterile antiseptic solutions or germicides, I am pleased to explain that until about fifteen years ago frequent irrigation was the rule, but since that time I have resorted to neither irrigations nor the application of germicides in the field of operation within the mouth. I examine the parts once daily and remove all sutures (silk or horsehair), silver wire and lead plates from the palate on the ninth to the eleventh day. The cutaneous sutures, as well as those on the labial surface of the mucous membrane, are kept free from any crust formations by the application of sterile ointment. On the third day we begin to remove the sutures from the skin, expecting to have them all out not later than the fifth day, with the exception of those on the under-surface of the lip, which are removed on the eighth day.

In closing, permit me to thank the Society for the compliment extended; I sincerely appreciate the opportunity of appearing before you. In addition, I desire to make a single statement that I feel certain will not be misunderstood, namely, it is my opinion that every physician who is granted the privilege of directing parents in the care of their children has not only a great obligation but a direct responsibility. And this load is especially heavy if the child in question is not physically fit, for on the physician's advice often rests, to a greater degree than he knows, the opportunity of being the determining factor in seeing to it that the congenitally defective infant is transformed into a healthy and normal-appearing child. However, I realize that if there is any one group of physicians who do not need this admonition it is the pediatricians, for by the selection of their specialty they have proved their great interest and their worthiness of the trust that the parents of these children place in them. Yet I ask that each should be ever mindful that a congenitally defective infant should be considered his special professional responsibility.

Dr. Thomas L. Grisamore: The habits that have been spoken of in the discussion constitute a problem for any one who handles children. Thumb-sucking, the use of pacifiers and labial and sleeping habits all have a tendency to produce deformity. In fact, any abnormal pressure, even though it be slight, if continued for any great length of time is liable to produce deformity. The thumb-sucking that was particularly referred to in the discussion should be stopped as early as possible. In most cases, as the child grows older there seems to be greater difficulty in breaking the habit. In the majority of cases, if some slight appliance can be placed in the mouth that will prevent the child from producing a vacuum while sucking the thumb the habit will be discontinued.

Sleeping habits in some cases cause malformation of the dental arches and the face. Many of these habits are well developed, and much of the damage is

done before the child reaches the orthodontist. There can be no doubt of the fact that if the pediatrician and dentist cooperate in handling these young children a great many of the abnormalities of the teeth and deformities of the face will be prevented.

I wish to take this occasion to thank Dr. Logan for suggesting my name to the program committee, and I am pleased that this splendid audience of physicians has shown so great an interest in a strictly dental subject.

Regular Meeting, Feb. 18, 1936

ARTHUR H. PARMELEE, M.D., *Presiding*

THE RAW APPLE TREATMENT FOR DIARRHEA IN PEDIATRIC PRACTICE. DR. MAXWELL P. BOROVSKY.

A survey of the American literature reveals few articles on the raw apple diet for diarrhea in children, but the European publications since 1930 contain about sixty papers on this subject in practically every foreign language, principally in German.

Heisler, a practitioner in Königsfeld, Schwarzwald, used raw apples in the treatment of diarrhea for twenty years before he published his results in 1929.

Moro's reports of the use of the raw apple diet in his clinic are directly responsible for the popularization of this treatment. In practically every type of diarrhea the clinical observations were as follows: The stools were normal in two days, frequently in one day, after the treatment was begun, while vomiting ceased, the temperature dropped and abdominal cramps and tenesmus disappeared.

I have never found any one form of treatment that so simply and uniformly corrected a diarrheal tendency. Patients with acute conditions show the best results. In no instance has an aggravation of the condition of the bowel as a result of this therapy been encountered.

The treatment is simple, consisting merely of feeding the child scraped raw apple in varying amounts, depending on the patient's size and age. For young infants and children one or two tablespoonfuls every two or three hours is the average dose, and weak tea is allowed freely. All other foods are prohibited during the interval of treatment, from twenty-four to forty-eight hours. When the condition of the bowel has been corrected, well cooked cereals and properly modified milk may be offered, with a gradual return to the normal diet.

The beneficial effect of the raw apple is probably due to its adsorptive power and its detoxifying action. This fruit adsorbs and enmeshes the bacteria, mucus and toxins from the mucous membrane of the bowel. The cellulose, pectins, malic acid and tannates are all considered of great value.

Twenty-three cases of enteral and parenteral diarrhea are reported, in which uniformly good results were obtained within from fourteen to forty-eight hours after the institution of this treatment. The age of the patients varied from a few days to 8 years. The youngest patient was 15 days old, and the smallest weighed 4 pounds and 12 ounces (2,154.5 Gm.).

This treatment is deserving of more general consideration.

DISCUSSION

DR. BENGT L. K. HAMILTON: Has this treatment ever been used in chronic ulcerative colitis?

DR. LEONARD HARRIS: I have observed a number of cases of diarrhea and have used this treatment. In my experience I have found that it makes little difference whether the apple is peeled or not or whether it is used raw or cooked.

One important point is to omit milk for several days and to give plenty of apple when it is started. If one prepares the apple as a soft mush with tea it can be fed through the nipple, and babies take it readily.

Dr. Gerard N. Krost: Mention has been made of cooking the apples. Some apples are sweet, and some are very tart. Which is preferable? Is there any clinical rule as to how much apple shall be given in a day? Dr. Borovsky mentioned the quantity in grams, but I think that the size of the child would make a great deal of difference.

Dr. Mandel L. Spivek: This treatment was tried experimentally at the Cook County Hospital about four years ago. Twenty-five children with diarrhea were put on apple treatment, and twenty-five children with a similar condition were used as controls. All the patients were acutely ill, much more so than is normally found in practice. The children on the apple diet had formed stools soon after the treatment was started, but they were not stools in the ordinary sense, being brown or brown-green masses of apple. All the elements that one would expect in a diarrheal stool, namely, pus, blood and mucus, were present and intimately mixed with the apples. Statistically, those receiving the apple treatment were discharged one tenth of a day sooner than the controls.

Dr. Arthur H. Parmelee: My experience with this treatment in diarrhea has been confined to perhaps half a dozen cases in the hospital. The striking thing to me is that the stools do improve. They become semiformed, but, as Dr. Spivek said, they are not stools but look like apple butter. Also, the curve for weight shows a decided drop; a loss of 1 or 1½ pounds (0.5 or 0.7 Kg.) in a year old child in twenty-four hours was not unusual. My associates and I used the treatment for twenty-four or forty-eight hours at the most and then started feeding buttermilk immediately. The change in the stools and the rapid drop in weight were the most striking features that I noticed in connection with the treatment.

Dr. Maxwell P. Borovsky: In reference to ulcerative colitis, I mentioned one case that my associates and I observed in which there was symptomatic improvement. We did not expect to cure the patient but thought that there might be an aggravation of the trouble. The child was relieved of the tenesmus and marked abdominal discomfort. When he was taken off the apple treatment the soft, bloody stools again developed.

As to cooking the apple, I mentioned that some physicians have boiled the apple, peeling and all, and found the results satisfactory. Most physicians use the raw scraped apple.

As to the type of apple, Heisler has used green apples so sour that they caused puckering of the mouth, so the type does not seem to matter much. As I stated, the Germans have prepared a powdered apple in order to have a uniform type. In some places apples are scarce during some months of the year.

Dr. Harris mentioned using apple with tea; that can readily be done.

As to the amount, I suppose that one can safely give a tablespoonful every two hours. I think that one cannot very well overfeed patients with diarrhea, for they will not take that much apple. The diet does serve a good purpose.

Dr. Spivek's remarks make it seem that this treatment has merits. It is certainly better to bring about improvement with such a method than with medication.

It is true that there is a marked loss of weight, but the aim is to quiet the bowel and increase tolerance for food that will finally permit the building up of the child.

Further Studies on the Metabolism of Cevitamic Acid in Infantile Scurvy.

Dr. Arthur F. Abt and Chester J. Farmer, Ph.D. (by invitation).

A macro-method and a micro-method for the chemical determination of reduced cevitamic acid in the blood plasma have been described.

Values for the reduced cevitamic acid content of the blood plasma have been enumerated for infants and children on diets adequate in vitamin C and have been compared with determinations of the capillary skin resistance.

No correlation between age and the cevitamic acid content of the blood plasma could be demonstrated. Our results fail to substantiate synthesis of cevitamic

acid by young infants. Congenital scurvy is possible. The cevitamic acid content of blood plasma is directly variable with the vitamin C content of the diet. Determinations of the cevitamic acid of the blood plasma are an accurate index for the detection of subclinical scurvy. Values for the cevitamic acid of the blood plasma are a better index in healing scurvy than the capillary skin resistance or serial roentgenograms. The values for the blood plasma show a rapid rise in cases of healing scurvy, and a single determination on the blood is more informative than a determination on the urine.

With the aid of the micro-method hourly the results of determinations on the blood may be plotted.

DISCUSSION

DR. FRED O. TONNEY: This is a tremendously interesting presentation. Scurvy! As old as the history of man—a disease of armies and prisons, once known as mariner's disease because it appeared on long sea voyages.

I am reminded of Count von Luckner and his crew on the German raider in the World War. His description of scurvy is a classic—the frosty white gums with protruding teeth "like a wild animal's" (he wrote), painful, bleeding (and nothing but hardtack to eat); the blue-black hemorrhages under the skin, and the joints so greatly swollen that the trousers had to be slit in order to enable the men to crawl about the deck. Yet whenever they could land somewhere and get fresh green foods—even bananas—miraculously, the disease disappeared.

The incredible thing to me is that here in Chicago, where fresh fruits and vegetables are always available the year round, one should still have to deal with scurvy.

Again, it is most interesting to see the clinical picture reduced to mathematical precision in the tables shown tonight. Personally, I did not know that scurvy was still a problem here, and I am wondering whether anything can be done about it from the standpoint of public health.

DR. H. G. PONCHER: The microchemical technic for the determination of cevitamic acid in the blood proposed by Farmer and Abt will be a great aid in studying cases. My associates and I have resorted to study of the twenty-four hour output of urine because the previous methods necessitated venipuncture at frequent intervals. Daily study of the excretion of cevitamic acid in the urine seemed advisable from another standpoint. If proper vitamin C balance depended on saturation of tissues and if only after saturation of tissues would the level of cevitamic acid be raised in the twenty-four hour output of urine, it would appear that study of levels of the twenty-four hour urinary excretion might give valuable information. I should like to ask Dr. Abt, therefore, whether he believes that the level of cevitamic acid in the blood rises before tissue saturation and whether cevitamic acid shows in the blood before it becomes apparent in the twenty-four hour urinary excretion.

Practically, the microchemical technic on blood is easier to prosecute than examination of twenty-four hour specimens of urine, because of difficulty in collecting the specimens and the necessity of hospitalizing the patient in the latter procedure. However, it would be important to know the relative value of these methods in studying the fundamental factors governing the cevitamic acid balance. Tests of tolerance to cevitamic acid will probably throw some light on this point.

Also, if tissue saturation be the therapeutic objective, might it not be desirable to give larger doses at first to produce tissue saturation and then drop to the maintenance dose? It would seem that this might conceivably speed up the period of recovery. There is apparently no danger in larger doses, as my associates and I have given daily injections of 1,000 mg. of cevitamic acid to guinea-pigs, without untoward symptoms following, and have used doses up to 500 mg. daily in infants.

DR. ARTHUR F. ABT: From our experience to date, my associates and I feel that the level (in milligrams per hundred cubic centimeters) of reduced cevitamic acid in a single sample of blood taken while the patient is fasting is indicative of the tissue saturation. Curves for tolerance of the blood to cevitamic acid and

values for the twenty-four hour urinary excretion bear out this opinion. In other words, if one wishes to investigate tissue saturation to determine whether there is a great deal of subclinical scurvy, and whether many children in any region are not taking sufficient vitamin C, a single determination on the blood will give the desired information simply. There is now a technic by which, instead of puncturing a vein, one may take a few drops of blood from the finger or heel, which we feel is a much simpler method than taking twenty-four hour specimens of urine.

Posture in the Etiology, Prophylaxis and Treatment of Diseases of the Lung. Dr. Jerome R. Head (by invitation).

Man's assumption of the erect posture while waking and the supine posture while sleeping has resulted in serious distortions of the fundamental bodily architecture and in serious handicaps to function. The ailments attributable to them in the skeletomuscular, genito-urinary and gastro-intestinal systems have been long recognized and adequately considered, but their importance in the etiology of diseases of the respiratory system still needs emphasis.

In the normal horizontal prone position secretions from the lungs and trachea tend to drain downward and forward to the pharynx, and those from the pharynx, mouth and nose tend to pass outward through the mouth and nostrils. In the erect and supine positions the reverse is true. Gravity favors the entrance of foreign material and secretions into the trachea and lungs and the stagnation of secretions formed in situ.

It formerly was believed that the reflex closure of the glottis and the ciliary action of the respiratory epithelium and the cough reflex were wholly efficient in protecting the lower respiratory system from gross contamination. Recent experiences with iodized poppy-seed oil 40 per cent and with the injection of other substances into the bronchial tree have shown that this belief is unfounded and that the whole protective mechanism can be easily penetrated.

There is much experimental and clinical evidence that gravity and gross contamination are important factors in the etiology of pulmonary disease. Lobar pneumonia can be produced only by the intratracheal injection of relatively large volumes of bacterial suspension. Pulmonary abscess can be produced only in the same manner. Clinically, it is of the greatest significance that a high percentage of all nontuberculous, pulmonary and bronchial diseases occur in the lower and posterior portions of the lungs. It is equally significant that in animals the favorite site is in the anterior portions of the lungs.

If these facts are true, their implications in prophylaxis and treatment are obvious. One cannot expect that man will return to walking on all fours, but it is reasonable to advise persons to sleep on the abdomens and when disease does attack the lungs to assume a position which will favor the elimination of secretions.

DISCUSSION

Dr. A. H. Andrews Jr.: It was a pleasure to hear this paper. I should like to ask Dr. Head about the relationship of posture to physiologic atelectasis, and their relation to pulmonary conditions. There has recently been some work by Yates in which he studied the ciliary pathways of the nose and pharynx. He reported that by ciliary activity dyes placed in the nose pass down the lateral walls of the pharynx. In one case he traced the dye down to the posterior laryngeal commissure and then into the trachea. In this case the patient had laryngitis which was definitely secondary to sinusitis, so there are other factors to be considered in addition to posture.

Dr. Eugene T. McEnery: I have always felt that children who slept on their faces were less subject to bronchitis and otitis media than others, because drainage was better. On the other hand, I wonder whether Dr. Head has con-

sidered this point: A child raises the hands toward the face when sleeping on the abdomen and, frequently, because of the proximity of the hands to the mouth becomes a thumb-sucker.

In bronchiectasis, postural drainage is very good in treatment. One thing should be stressed: It is not sufficient for the child to lie on the face, but the buttocks should be elevated to such a degree that good drainage is afforded.

DR. JOSEPH BRENNEMANN: Dr. Head has presented an interesting and stimulating paper. My associates and I at the Children's Memorial Hospital have been much interested in both the etiology and the treatment of many of the conditions that he has touched on. It may interest Dr. Head that I cannot recall having observed a single case of postoperative massive collapse of the lung. We are looking for such cases and would recognize the condition if it occurred. On the other hand, we observe a large number of cases of conditions apparently due to some inflammatory or mechanical condition, in the etiology of which posture may play a greater part than we know. For example, we have found massive collapse of a lower lobe of the lung in patients with acrodynia, pseudohypertrophic muscular dystrophy and postdiphtheritic paralysis. One can readily assume that lying on one side, thus interfering with an already weakened respiratory mechanism, may lead to stagnation of secretions in the compressed dependent portion, with resulting atelectasis, especially in the presence of a bronchial inflammation with increased secretion. It seems not unlikely that posture, a prolonged constrained position, may play a part even if there is not a generally weakened respiratory musculature. This, at least, carries with it a therapeutic, notably, prophylactic, suggestion.

DR. HARRY H. BOYLE: Would Dr. Head apply postural drainage in cases of nasal infection and other infections of the upper respiratory tract?

Book Reviews

The Bacteriology of Typhoid, Salmonella and Dysentery Infections and Carrier States. By Dr. Leon C. Havens. Price, $1.75. Pp. 158, with 6 illustrations. New York: Commonwealth Fund, 1935.

This small volume should prove valuable to any one interested in the group of enteric infections caused by the typhoid Salmonella and dysentery groups of bacilli. Although Salmonella and the dysentery bacilli are rather widespread, their importance as pathogens has been so questioned by bacteriologists and physicians alike that their true importance has not been generally accepted. In the past few years more attention has been given the typhoid, Salmonella and dysentery groups of bacilli because of numerous epidemics of diarrhea in children and adults throughout the United States.

Dr. Leon C. Haven was director of laboratories of the Alabama Department of Public Health and had made special studies of these organisms for many years. Before his death he had about finished the first draft of his book, and his friend, Dr. Kenneth Maxcy, professor of preventive medicine at the University of Virginia, has ably edited the work. There is a foreword by Dr. Wilson G. Smillie, professor of public health administration, Harvard University.

There is a summary at the end of eight of the thirteen chapters. The author presents a clear discussion of antigens and their importance in these groups, the methods of culture of stools, the choice of culture mediums, the cultural and serologic identification of intestinal pathogens, the problem of typhoid carriers and special consideration of the typhoid, Salmonella and dysentery groups of bacilli.

This well written manual should be of special value to the public health official, epidemiologist and laboratory technician, as well as to any physician interested in a better understanding of the typhoid, Salmonella and dysentery groups.

The Bacteriology of Milk. By G. S. Wilson. Assisted by R. S. Twigg, R. C. Wright, C. B. Hendry, M. P. Cowell and J. Maier. Price, 7 shillings, sixpence. Pp. 392, with 166 tables and 29 illustrations. London: His Majesty's Stationery Office, 1935.

This book is published by the Medical Research Council and takes up in detail the technic of the tests used in the bacteriologic grading of milk and the interpretation of the tests used in this grading.

The work contains an immense amount of detail. The general conclusions are that all milk should be tested after standing from twelve to eighteen hours at atmospheric temperature. Cleanliness should be judged not by tests on one or two samples but by tests on samples taken frequently and regularly throughout the year. The sediment test, the leukocyte count, determination of the titrable acidity, estimation of the hydrogen ion concentration, the increase in acidity, the bromthymolsulfonphthalein (bromthymol blue) test, the keeping quality test and laboratory pasteurization cannot be regarded as suitable for the routine grading of milk. The test for coliform bacilli is of value only for freshly pasteurized milk. The plate count test is severely criticized. The Breed smear test is good, but the modified methylthionine chloride (methylene blue) test, which is fully described, is the most inexpensive procedure and gives the smallest percentage of error.

No matter what test is used, no attempt should be made to divide milk into more than three or four classes. From the point of view of public health, only two classes are necessary: (*a*) milk that is suitable for human consumption and (*b*) milk that is not suitable for human consumption.

This is an excellent work for any one interested in the subject. A large bibliography is given.

Sífilis congénita en el niño. By Calixto Torres Umaña. Paper. Pp. 312, with 14 photographs. Bogota: Editorial Cromos, 1935.

This is a study of congenital syphilis in Bogota, Colombia. The immensity of this problem in Colombia can best be realized by considering the author's figures for the frequency. He estimates this at 6 per cent in cases observed in private practice and at 25 per cent in cases observed in clinics. The discussion is taken up in the usual form of statistics, frequency, pathology, laboratory tests, symptoms, prognosis and treatment. All these subjects are presented in a systematic manner. It is of especial interest to one working with these problems to read of the many and varied forms that the symptoms of florid congenital syphilis may take. Of particular interest is the discussion of the relation of rickets and toxic infections such as syphilis. In France, Marfan found 40 per cent of children in the Paris Hospital between the ages of 3 months and 3 years to be rachitic. Only 4 per cent of the children in the author's practice were rachitic, and of these only 3 per cent were syphilitic. This may be an answer to Marfan's argument of the association of the two diseases. In Colombia there is very little rickets but much congenital syphilis.

The methods of treatment are similar to those in use in this country. Although the work is in Spanish, it is so well written that a reader with only a casual knowledge of this language can follow it with ease and will find it both interesting and profitable reading.

Common Contagious Diseases. By Philip Moen Stimson. Price, $4. pages, with 53 engravings and 3 plates. Philadelphia, Lea & Febiger, 1936.

This is the second edition of this book. It has been revised and is completely up to date. In the opinion of the reviewer, this is a work which is peculiarly fitted for the use of general practitioners and especially those who are most interested in children.

The first chapters, "Principles of Contagion" and "Serum Reaction," are most enlightening, and the whole book is systematically prepared and clearly written. After the various contagious diseases are taken up, there is a chapter on "General Management of Contagious Diseases," including a discussion of the management of these diseases in hospitals.

A glossary is appended at the end of the book, and there is a tabulated summary of the common contagious diseases. The illustrations are clear and depict the conditions extremely well. On the whole, the book is well worth being in the possession of any physician.

A Diabetic Primer for Children. By Ella M. Coleman and Alfred E. Fischer, M.D. Pp. 42, with 4 illustrations. New York: Mount Sinai Hospital, 1935.

The authors have written a pamphlet in the second person which is intended for the child in the grade schools who cannot comprehend the usual primers on this subject. They have succeeded admirably in simplifying terminology.

The new classifications of fruits and vegetables recently accepted by the American Dietetic Association have been included. In the sample diets the fats and proteins remain about the same in amount, while the amount of carbohydrates is varied according to age. Each diet therefore has a considerable variation in the ketogenic-antiketogenic ratio. There is a good list of substitute foods. The food recipes are devised to appeal especially to the child.

The method of administration of insulin is explained in detail. The reaction to insulin is well described, so that the child should not be unduly alarmed but rather well educated and prepared to cope with it. In the description of approaching coma the authors fail to indicate the necessity for tests for the ketone bodies in the presence of illness or persistent increasing glycosuria.

The pamphlet is well adapted for the education of the diabetic child. It would seem that it could be improved by the use of larger type.

L'otite latente del lattante in pediatria. By Dr. Renato Canino. Pp. 95. Naples: Arti Grafiche La Nuovissima, 1935.

This book is based on experience with 1,885 babies in the Brefotrofio della Annunziata of Naples. Of these, 366 had severe disease syndromes, and of this number 26.2 per cent had an aural condition. The monograph takes up carefully the etiology and pathologic features of the condition and discusses at some length the relation of the latent otitis media to the various conditions encountered. In the opinion of Canino, in most instances otitis media presents a severe complication and in some may be regarded as a distinct causal factor, especially with respect to nutritional disturbances. In 96 cases of diarrheal conditions, 47.9 per cent of the patients died. The author advises in the way of treatment early paracentesis, which should be repeated, if necessary, but thinks that it is rarely wise to do an antrotomy until after other measures have failed. He emphasizes the dangers of this condition at this age and throughout the book stresses the difficulty of diagnosis of the condition because of the uncertainty of examinations of the ear in infancy.

For Stutterers. By Smiley Blanton, M.D., and Margaret Gray Blanton. Price, $2. Pp. 186. New York: D. Appleton-Century Company, Inc., 1936.

This book is written for lay readers, as its title implies. There is an understandable discussion of medical psychology, with chief emphasis on the freudian conception. The authors' theory of stuttering and their ideas of treatment are based largely on this. They arrive at somewhat the same conclusions stated by other authors in regard to the correction of personality difficulties and maladjustments, the removal of coercions of parents and teachers and the lessening of social stresses as a prerequisite for the relief of these patients. Some authorities will consider the exposition as narrow and based on incomplete and not fully proved theory.

Stutterers will get from this volume some helpful understanding of their own difficulties.

Morbilidad y mortalidad en la edad pre-escolar. By G. L. Bayley Bustamante. Paper. Pp. 150, with 11 graphs and 87 tables. Buenos Aires: Aniceto Lopez, 1935.

This is a detailed study of children of preschool age (from 2 to 6 years) in Argentina for the years 1930 to 1933. The statistics for both the morbidity and the mortality are given and are further divided into statistics on the various causes, on sex and on age incidence. Although the book is in Spanish, the tables and graphs are so clear that any one can easily read them. Much work has been put into this study, and the form and material give ample evidence of this. It is an excellent work for any one interested in this branch of pediatrics, and it is gratifying to see that pediatricians of other countries are taking an interest in the child of preschool age.

Annual Report of the Surgeon General of the Public Health Service of the United States. Secretary of Treasury Department. Cloth. Price, 75 cents. Pp. 158, with 2 illustrations. Washington, D. C.: United States Government Printing Office, 1935.

Most of this report is presented in a readable and interesting form. The foreword presents a summary of world health conditions and of health conditions in the United States. No quarantinable disease was introduced into the United States from abroad during the year, and the prevention of spread of disease through interstate traffic was, for the most part, successful. The report of each division, such as those of scientific research, quarantine, marine hospitals, venereal disease, mental hygiene, personnel, and accounts, are given separately.

Directory of Pediatric Societies *

INTERNATIONAL

International Association of Preventive Pediatrics

President: Professor G. Scheltema, 6a Heereplein, Groningen, The Netherlands.
Secretary: Dr. Daniel Oltramare, 15 Rue Lévrier, Geneva, Switzerland.

FOREIGN

British Paediatric Association

President: Dr. Leonard Findlay, 14, Wimpole St., London, W. 1.
Secretary: Dr. Arthur G. Maitland Jones, 31 A, Weymouth St., London, W. 1.

Royal Society of Medicine, Section for the Study of Disease in Children

President: Sir Lancelot Barrington-Ward, 85 Harley St., London, W. 1.
Secretary: Dr. K. H. Tallerman, 122 Harley St., London, W. 1.
Place: 1, Wimpole St., London. Time: Fourth Friday of each month, 4:30 p. m.

Paediatrický Spolok na Slovensku

President: Dr. A. J. Chura, Lazaretská 6, Bratislava, Czechoslovakia.
Secretary: Dr. S. Limbacher, Dlhá 2, Bratislava, Czechoslovakia.
Place: Pediatric Clinic, University, Bratislava. Time: Six times a year.

Sociedad Cubana de Pediatría

President: Prof. Félix Hurtado Galtés, Carlos III, 7, Havana.
Secretary: Dr. Carlos Hernández Miyares, San Lazaro 370, Havana.
Place: Cátedra de Clínica Infantil, Hospital Mercedes, Havana. Time: Third Thursday of every month.

NATIONAL

American Medical Association, Scientific Assembly, Section on Diseases of Children

Chairman: Dr. Ralph M. Tyson, 255 S. 17th St., Philadelphia.
Secretary: Dr. Albert D. Kaiser, 16 N. Goodman St., Rochester, N. Y.
Place: Atlantic City, N. J.

American Academy of Pediatrics

President: Dr. Laurence R. DeBuys, 1417 Delachaise St., New Orleans, La.
Secretary: Dr. C. G. Grulee, 636 Church St., Evanston, Ill.
Region II. Place: Baltimore. Time: Nov. 16 and 17, 1936.
Region III. Place: Cincinnati. Time: Nov. 20 and 21, 1936.
Region: IV. Place: San Francisco. Time: Oct. 22-24, 1936.

American Hospital Association, Children's Hospital Section

President: Mr. Robert Witham, Children's Hospital, Denver.
Secretary: Miss Agnes O'Roke, Kosair Crippled Children's Hospital, Louisville, Ky.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

American Pediatric Society

President: Dr. Fritz B. Talbot, 270 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Hugh McCulloch, 325 N. Euclid St., St. Louis.
Place: Bolton Landing, N. Y. Time: June 11-13, 1936.

Canadian Society for the Study of Diseases of Children

President: Dr. A. P. Hart, 1844 Bloor St., W., Toronto, Ont.
Secretary-Treasurer: Dr. Pearl Summerfeldt, Hospital for Sick Children, Toronto, Ont.
Place: Bolton Landing, N. Y. Time: June 11-13, 1936.

Society for Pediatric Research

President: Dr. Francis Scott Smyth, University of California Medical School, San Francisco.
Secretary: Dr. A. A. Weech, 3975 Broadway, New York.

SECTIONAL

North Pacific Pediatric Society

President: Dr. L. Howard Smith, 1020 S. W. Taylor St., Portland, Ore.
Secretary: Dr. Morris Bridgeman, Children's Clinic, Portland, Ore.

Southern Medical Association, Section on Pediatrics

Chairman: Dr. Madison Hines Roberts, 104 Ponce de Leon Ave., N. E., Atlanta, Ga.
Secretary: Dr. Hughes Kennedy Jr., Highland Plaza Apts., Birmingham, Ala.

STATE

Alabama Pediatric Society

President: Dr. J. W. Britton, 931 Noble St., Anniston.
Secretary-Treasurer: Dr. Wallace Clyde, Employees' Hospital, Fairfield.

Arkansas State Pediatric Association

Chairman: Dr. Morgan Smith, 625 Beech St., Little Rock.
Secretary: Dr. Madeline A. M. Melson, 2111 N. Spruce St., Little Rock.

California State Medical Society, Section on Pediatrics

Chairman: Dr. S. J. McClendon, 2001 Fourth Ave., San Diego.
Secretary: Dr. Edward B. Shaw, 384 Post St., San Francisco.
Place: Coronado. Time: May 25-28, 1936.

Florida State Pediatric Society

President: Dr. Luther W. Holloway, 117 W. Duval St., Jacksonville.
Secretary: Dr. Warren W. Quillian, 227 Avenue Aragon, Miami.
Place: Concurrent with state association meeting at time of convention.

Georgia Pediatric Society

President: Dr. William Willis Anderson, 478 Peachtree St., Atlanta.
Secretary-Treasurer: Dr. Roger W. Dickson, 33 Ponce de Leon Ave., N. E., Atlanta.
Place: Savannah. Time: June 1936.

Indiana State Pediatric Society

President: Dr. Milo K. Miller, 122 N. Lafayette Blvd., South Bend.
Secretary-Treasurer: Dr. Mathew Winters, 23 E. Ohio St., Indianapolis.
Time: Two meetings a year.

Louisiana State Pediatric Society

President: Dr. Charles James Bloom, 3439 Prytania St., New Orleans.
Secretary-Treasurer: Dr. J. A. Crawford, 834 Ryan St., Lake Charles.

Medical Society of State of New York, Section on Pediatrics

Chairman: Dr. George M. Retan, 459 James St., Syracuse.
Secretary: Dr. John D. Craig, 33 E. 61st St., New York.

Michigan State Medical Society, Pediatric Section

Chairman: Dr. Edgar E. Martmer, 749 David Whitney Bldg., Detroit.
Secretary: Dr. C. R. Dengler, 131 W. Michigan Ave., Jackson.

Mississippi State Pediatric Society

President: Dr. R. E. Wilson, 205 S. Harvey St., Greenville.
Secretary-Treasurer: Dr. Guy C. Jarratt, 920 Crawford St., Vicksburg.
Place: To be designated by Society. Time: Yearly.

Nebraska Pediatric Society

President: Dr. J. A. Henske, 1614 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. H. Murphy, 915 Medical Arts Bldg., Omaha.
Place: As announced by committee. Time: Third Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

North Carolina Pediatric Society

President: Dr. Leroy J. Butler, 405 N. Spring St., Winston-Salem.
Secretary: Dr. C. A. Street, 405 N. Spring St., Winston-Salem.
Place: Roaring Gap. Time: August 1936.

Oklahoma State Pediatric Society

President: Dr. Julian Feild, 610 S. Monroe St., Enid.
Secretary: Dr. B. H. Nicholson, 301 N. W. Twelfth St., Oklahoma City.

Pennsylvania Medical Society, Pediatric Section

Chairman: Dr. Joseph Stokes, Children's Hospital, Philadelphia.
Secretary: Dr. Theodore O. Elterich, 310 Medical Arts Bldg., Pittsburgh.

South Carolina Pediatric Society

President: Dr. Leland B. Salters, 317 S. Warley St., Florence.
Secretary-Treasurer: Dr. D. Lesesne Smith Jr., 187 Oakland Ave., Spartanburg.

Tennessee State Pediatric Society

President: Dr. Joe T. Smith, 702 Medical Arts Bldg., Knoxville.
Secretary: Dr. Kinsey M. Buck, 1193 Madison Ave., Memphis.

Texas Pediatric Society

President: Dr. C. O. Terrell, 813 Medical Arts Bldg., Fort Worth.
Secretary-Treasurer: Dr. F. H. Lancaster, 1507 Medical Arts Bldg., Houston.

Virginia Pediatric Society

President: Dr. Basil B. Jones, 1028 W. Franklin St., Richmond.
Secretary: Dr. W. A. McGee, 616 W. Grace St., Richmond.

West Virginia State Medical Society, Section on Pediatrics

President: Dr. John T. Thornton, 58, 16th St., Wheeling.
Secretary: Dr. Claude L. Holland, 400 Locust Ave., Fairmont.

LOCAL

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. J. D. Pilcher, City Hospital, Cleveland.
Secretary: Dr. William M. Champion, 2323 Prospect Ave., Cleveland.
Place: Cleveland Medical Library Bldg. Time: October, December, February and April.

Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. T. B. Verner, 170 St. George St., Toronto.
Secretary: Dr. D. V. Hutton, 170 St. George St., Toronto.

Bronx Pediatric Society

President: Dr. Harry D. Pasachoff, 1560 Grand Concourse, New York.
Secretary: Dr. Samuel Tripler, 1555 Grand Concourse, New York.
Place: Concourse Plaza Hotel, 161st St. and Grand Concourse. Time: Every second Wednesday of each month, except June, July, August and September.

Brooklyn Academy of Pediatrics

President: Dr. Thurman B. Givan, 115 Remson St., Brooklyn.
Secretary: Dr. John A. Monfort, 861 Carroll St., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Fourth Wednesday of each month, except May, June, July, August and September.

Central New York Pediatric Club

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various cities in New York. Time: Third Thursday of April and September.

Chicago Pediatric Society

President: Dr. A. Levinson, 30 N. Michigan Ave., Chicago.
Secretary: Dr. James H. Wallace, 715 Lake St., Oak Park, Ill.
Place: Children's Memorial Hospital, 710 Fullerton Ave. Time: Third Tuesday of each month.

Dallas Pediatric Society

President: Dr. I. T. Nesbitt, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. John E. Ashby, 614 Medical Arts Bldg., Dallas, Texas.
Place: Bradford Memorial Hospital. Time: 1 p. m., first and third Saturdays of each month.

Detroit Pediatric Society

President: Dr. Edgar E. Martmer, 749 David Whitney Bldg., Detroit.
Secretary: Dr. W. J. Scott, 5140 Second Ave., Detroit.
Place: Wayne County Medical Society. Time: 8:30 p. m., first Wednesday of each month.

Fulton County Medical Society, Pediatric Section (Atlanta, Ga.)

Chairman: Dr. Joseph Yampolsky, 478 Peachtree St., Atlanta.
Secretary: Dr. T. I. Willingham, 384 Peachtree St., Atlanta.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: Second Thursday of each month from October to April, 8 p. m.

Harris County Pediatric Society

President: Dr. E. O. Fitch, 3717 Main St., Houston, Texas.
Secretary-Treasurer: Dr. Fred Ames, Medical Arts Bldg., Houston, Texas.
Place: Houston Club. Time: Third Monday of each month.

HEZEKIAH BEARDSLEY PEDIATRIC CLUB OF CONNECTICUT

President: Dr. H. W. Brayton, 179 Allyn St., Hartford.
Secretary: Dr. Ernest Caulfield, 683 Asylum Ave., Hartford.

INTERMOUNTAIN PEDIATRIC SOCIETY

President: Eugene Smith, 385 Twenty-Fourth St., Ogden, Utah.
Secretary-Treasurer: Dr. W. C. Cheney, 837 Boston Bldg., Salt Lake City, Utah.
Place: Salt Lake City, General Hospital. Time: First Thursday of each month, 8 p. m.

KANSAS CITY (MISSOURI) PEDIATRIC SOCIETY

President: Dr. Harry M. Gilkey, 1103 Grand Ave., Kansas City.
Secretary: Dr. S. A. Stadler, 6133 Brookside Blvd., Kansas City.
Place: Kansas City General Hospital. Time: On call.

LOS ANGELES COUNTY MEDICAL ASSOCIATION, PEDIATRIC SECTION

President: Dr. George P. Laton, 1136 W. 6th St.
Secretary-Treasurer: Dr. Edwin F. Patton, 3875 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Headquarters, 1925 Wilshire Blvd.
Time: Second Monday of February, April, June, October and December.

MEDICAL SOCIETY OF THE COUNTY OF KINGS AND THE ACADEMY OF MEDICINE OF BROOKLYN, PEDIATRIC SECTION

President: Dr. Harry Apfel, 988 Park Pl., Brooklyn.
Secretary: Dr. Max Palinsky, 1270, 45th St., Brooklyn.
Place: 1313 Bedford Ave., Brooklyn. Time: Third Wednesday of each month, October to May.

MEDICAL SOCIETY OF THE COUNTY OF QUEENS, INC., SECTION ON PEDIATRICS

Chairman: Dr. W. C. A. Steffen, 42 Union St., Flushing, N. Y.
Secretary-Treasurer: Dr. Edith A. Mittell, 144, 38th Ave., Flushing, N. Y.
Place: Queens County Medical Bldg., Forest Hills, N. Y. Time: Third Monday of October, January, March and May.

MEMPHIS PEDIATRIC SOCIETY

President: Dr. W. D. Mims, 1042 Madison Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. W. R. Graves, 1193 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month, except June, July and August, 8 p. m.

MILWAUKEE PEDIATRIC SOCIETY

President: Dr. J. Gurney Taylor, 324 E. Wisconsin St., Milwaukee.
Secretary: Dr. H. B. Miner, 425 E. Wisconsin Ave., Milwaukee.
Place: Children's Hospital. Time: Second Wednesday of each alternate month, beginning with February.

NEW ENGLAND PEDIATRIC SOCIETY

President: Dr. Paul Emerson, 319 Longwood Ave., Boston.
Secretary-Treasurer: Dr. Henry E. Gallup, 66 Commonwealth Ave., Boston.
Place: Boston Medical Library. Time: Four meetings a year, occurring from September to May.

NEW YORK ACADEMY OF MEDICINE, SECTION OF PEDIATRICS

Chairman: Dr. Béla Schick, 17 E. 84th St., New York.
Secretary: Dr. Alexander T. Martin, 107 E. 85th St., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

Northwestern Pediatric Society

President: Dr. A. V. Stoesser, University Hospital, Minneapolis.
Secretary: Dr. Roger L. J. Kennedy, Mayo Clinic, Rochester, Minn.
Place: Minneapolis, St. Paul, Duluth and Rochester. Time: January, April, July and October.

Philadelphia Pediatric Society

President: Dr. H. H. Perlman, 1904 N. Franklin St., Philadelphia.
Secretary: Dr. James E. Bowman, 4091 Comly St., Philadelphia.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday of each month from October to June, inclusive.

Pittsburgh Pediatric Society

President: Dr. George J. Feldstein, 3401 5th Ave., Pittsburgh.
Secretary-Treasurer: Dr. E. R. McClusky, 3200 Fifth Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Tuesday, alternate months from October to April, inclusive.

Richmond Pediatric Society

President: Dr. W. E. Chapin, 1001 E. Clay St., Richmond, Va.
Secretary-Treasurer: Dr. Emily Gardner, 1100 W. Franklin St., Richmond, Va.
Place: Richmond Academy of Medicine, 1200 E. Clay St. Time: First Thursday of each month (except July, August and September).

Rochester Pediatric Society

President: Dr. F. W. Bush, 29 N. Goodman St., Rochester, N. Y.
Secretary: Dr. David Wolin, 16 N. Goodman St., Rochester, N. Y.
Place: Arranged by program committee. Time: Second Friday of each month.

Rocky Mountain Pediatric Society

President: Dr. Ralph Verploeg, 1850 Gilpin St., Denver.
Secretary: Dr. Max M. Ginsburg, Metropolitan Bldg., Denver.
Place: Children's Hospital, Denver. Time: First Saturday of each month from September to May, inclusive, 2:30 p. m.

St. Louis Pediatric Society

President: Dr. J. V. Cooke, St. Louis Children's Hospital, St. Louis.
Secretary-Treasurer: Dr. Katherine Bain, Beaumont Bldg., St. Louis.
Place: St. Louis Medical Society. Time: Third Friday of every month.

Seattle Pediatric Society

President: Dr. Arthur E. Wade, 1215, 4th Ave., Seattle.
Secretary: Dr. Walter B. Seelye, Cobb Bldg., Seattle.
Place: College Club. Time: Third Friday of each month, 6:30 p. m.

Southwestern Pediatric Society

President: Dr. Victor E. Stork, 1136 W. Sixth St., Los Angeles.
Secretary: Dr. Howard R. Cooder, 3875 Wilshire Blvd., Los Angeles.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Samuel Levin, 2501 W. Grand Blvd., Detroit.
Secretary: Dr. D. Murray Cowie, University of Michigan, Ann Arbor, Mich.

Westchester County Medical Society, Pediatric Section (New York)

President: Dr. Louis V. Waldron, 149 N. Broadway, Yonkers, N. Y.
Secretary-Treasurer: Dr. Fairfax Hall, 421 Huguenot St., New Rochelle, N. Y.
Place: Grasslands Hospital. Time: Third Thursday, Oct., Dec., Feb. and April.

INDEX TO VOLUME 51

The asterisk (*) preceding the page number indicates an original article. Subject entries are made for all articles. Author entries are made for original articles and society transactions. Biographies, Book Reviews, Obituaries and Society Transactions are indexed under the letters B, O and S, respectively.

Abdomen, acute, and appendicitis due to purpura, 1444
acute, and encephalomeningitis at beginning of pneumonia, 1195
acute conditions in scarlet fever, 418
enlargement, etiology of, 1195
pain in children, 173
pain, probable acrodynia characterized by syndrome of, 1467
surgery, 1228
teratoma, 1447
tumors; puncture biopsy as diagnostic measure in differentiation of solid tumors, 986
Abnormalities and Deformities: See also Monsters; Rhino-Encephalocele; Twins; and under names of diseases, organs and regions, as Anus, imperforate; Esophagus, atresia; Fingers and Toes, abnormalities; Foot, cleft; Heart, abnormalities; Microcephaly; etc.
amyoplasia congenita, 440
caused by prenatal lesions of striated muscle, 159
congenital, 409
disturbances in developmental functions of germ cells; case of anencephaly, 1460
enterogenous cysts and diverticula, *1126
maternal age at conception of congenitally malformed child; study based on 607 cases, *1007
multiple deformities in infant with adiposogenital syndrome, 909
multiple deformities in 1 family, 908
Abscesses: See also under names of organs and regions, as Liver, abscess; Lungs, abscess; etc.
multiple residual, with generalized erysipelas in 2 month old infant; pharyngolaryngeal abscess of pyriform sinus opened surgically by cervical route; recovery, 687
peritonsillar, incision and tonsillectomy in course of, 1220
retropharyngeal, with hemorrhage, 226
subphrenic, 711
subphrenic, in early infancy, 182
Abt, A. F.: Erythema annulare rheumaticum, 228
Letterer-Siwe's disease; splenohepatomegaly associated with widespread hyperplasia of nonlipoid-storing macrophages; discussion of so-called reticulo-endothelioses, *499
Metabolism of cevitamic acid in infantile scurvy, 1488
William Wadd; nursery mems and anecdotal biographies, *131
Acetonemia: See Blood, ketone bodies
Acetonuria: See Urine, ketone bodies
Achlorhydria: See Stomach, acidity
Achondroplasia confined to half the upper end of tibia; rapid evolution in 12 year old girl, 409
dyschondroplasia (Ollier's disease) with report of case, 681
in native of Tunis, 1192
Acid, Amino: See Amino-Acids
amino-acetic; creatinuria of infancy and childhood; normal variations; creatine tolerance tests and effect of amino-acetic acid in normal infants, *30
Ascorbic: See Acid, cevitamic
Cevitamic: See Scurvy; Vitamins, C
Acid—Continued
fatty, unsaturated fatty acid (vitamin F) deficiency, 1418
lactic acid skimmed milk soup, 1156
lactic; fermentation of milk by Mycotorulae isolated from intestines, 1155
Nucleic: See Nucleins
Acid-base equilibrium in dystrophic infants, 152
Acidosis and ketogenic diet; application to treatment of epilepsy and infections of urinary tract, 1207
of guanidine intoxication, 675
red corpuscles in, 1420
so-called acidosis attacks; plea for more accurate diagnosis, 1421
so-called, in childhood, 902
surgical importance of ketosis and cyclic vomiting, 678
Acrodynia, 212
probable case characterized by syndrome of abdominal pain, 1467
with quadriplegia, 211
Actinomycosis in childhood, 930
of lung, 1177, 1438
Acupuncture, Chinese method of, in treatment of nurslings with anorexia, 1423
Adams-Stokes' Disease: See Heart, block
Addis Sediment Count: See Urine, examination
Addison's disease treated by panirradiation, 1447
Adenitis: See under Lymph Nodes
Adenoids, complications and results of tonsil and adenoid operations, 460
Leishman-Donovan parasites in rhinopharynx, 1218
removal in scarlet fever; survey, 1433
treatment of adenoid septicemias by crushing vegetations with finger, 462
Adenoma of pituitary with reference to pituitary basophilism of Cushing, 949
Adenopathy: See under Lymph Nodes
Adiposity: See Obesity
Adiposogenital Syndrome: See Laurence-Biedl Syndrome; Pituitary Body
Adolescence, basal metabolism and energy exchange of adolescent girls, *801
fat and protein metabolism of adolescent girls, *1083
metabolism of adolescent girls; excretion of creatinine and creatine, *1268
ossification praecox, 1452
physiopsychology of puberty, 1151
precocious general development and obesity, 716
precocious puberty; gigantism, virilism, and pseudohermaphroditism, 951
precocious puberty in boy, 1190
precocious puberty; menstruation in 1 year old child with marked development of secondary sex characteristics, 1198
precocious puberty; specimens from cases of right-sided hemihypertrophy associated with pubertas praecox, 1429
precocious puberty with hydrocephalus following postbasic meningitis, 957
relation between basal metabolism and adolescent growth, *1014
Adoptions and the physician, 213
Adrenalin: See Suprarenal Preparations
Adrenals: See Suprarenals
Agglutination; Hanganatziu-Deicher reaction; hemagglutination, 1199
Agglutinins: See also Antigens and Antibodies; Blood, groups; Immunity; etc.
for sheep and rabbit erythrocytes in human sera, 673
sheep and rabbit cell agglutinins in horse serum sickness and infectious mononucleosis, 674

Agranulocytosis: See Angina, agranulocytic
Air studies at higher altitudes, 148
Akinesia: See Movements
Albers-Schönberg's Disease: See Osteosclerosis fragilis
Albumin: See Albuminuria; Protein
Albuminuria, 433. See also Retinitis, albuminuria
functional (orthostatic), 1448
Alcohol, effect on efficiency in sports, 1230
Aldrich, C. A.: Special report of American Board of Pediatrics, Inc., 389
Aldrich and McClure Test: See under Edema
Alimentary Intoxication: See Toxemia
Alkalosis: See also Acid-base equilibrium
red corpuscles in, 1420
Allergens: See Anaphylaxis and Allergy
Altitude: See also Climate
air studies at higher altitudes, 148
variations in cholesterol content of blood in children with pulmonary tuberculosis after sojourn at high altitude, 168
Aluminum as factor in different pathologic processes, 1452
salts, effect of feeding high amounts, 899
Amatruda, C. S.: Comparative study of 6 infant cretins under treatment; diagnostic aspects, 1236
Amebiasis, 943. See Liver, abscess, amebic acute, 922
American Board of Pediatrics, examinations, 897
physicians certified by, 145, 897, 1415
report of, 389
American Orthopsychiatric Association, annual session, 145
Amidopyrine, as cause of agranulocytosis, 1453
Amino-Acids in Blood: See Blood, amino-acids quantity in placenta, 909
Ammonia in Urine: See Urine, ammonia
Amniotic fluid; therapeutic value and effects of amniotin in gonorrheal vaginitis, 171
Amniotin: See Amniotic fluid
Amyloidosis and lipoid nephrosis in pulmonary tuberculosis, 936
case of amyloid kidney with subacute course simulating hypochloremic azotemia, 1448
Amyoplasia Congenita: See Joints, ankylosis; Muscles
Amyotonia: See Myatonia
Congenita: See Myotonia congenita
Anaphylaxis and Allergy: See also Asthma; Food, allergy; etc.
abdominal pain due to allergy, 173
acute anaphylactic shock following intracutaneous test for sensitivity to horse serum, 709
allergic child; actual status of this question, 182
allergic condition due to menotoxin, 221
allergic diseases in childhood, 941
allergic management of vasomotor rhinitis, 456
auto-injection of urine in, 184
bacterial allergy and immunization process, 672
bacterial allergy, diagnosis of, 711
circulating allergic antibody, 996
comparative yearly surveys of specific sensitizations in students of University of Michigan from 1930 to 1935, 994
diagnostic patch tests in determination of arsphenamine sensitization, 710
diphtheritic, focal reactions in, 692
eosinophilia in allergic conditions, 1451
facial and dental deformities due to perennial nasal allergy, 458
Food Allergy: See Food, allergy
histamine in relation to intradermal reaction to allergens in sensitized persons, 997
influence of acid or alkali on anaphylactic shock, 697
interpretation of early and delayed intradermal reactions in allergic persons, 996
Anaphylaxis and Allergy—Continued
metabolism, allergy and autonomic nervous system, 714
milk as cause of anaphylaxis, 1154
pathogenesis and management of allergy, 1242
rapid hyposensitization, 707
reagin content of blister fluid, 172
sensitization and intelligence quotient, 994
serum allergy; antigenic relationship between horse dander and horse serum sensitivity, 1183
serum sickness, clinical and experimental study, 425
sheep and rabbit cell agglutinins in horse serum sickness and infectious mononucleosis, 674
species nonspecific antigenic factor in mammalian serums, 1182
Andresen, M. I.: Values for red blood cells of average infants and children, *775
Andrews, E. T.: Pneumonia; bacteriologic study with special reference to clinical significance, *1277
Anemia, Agranulocytic: See Angina, agranulocytic
and deficiency disorders in relation to nonspecific colitis, 440
Cooley's, 1190
hemolytic, acute, 180
hyperchromic, in icterus gravis neonatorum, *104
iron deficiency as cause of, 1200
iron deficiency; its association with gastrointestinal disease, achlorhydria and hemorrhage, 948
iron metabolism in relation to nutritional anemia, 400
mechanism; physiologic anemia, 1181
milk as cause of, pathologic study, 947
pernicious; hematopoietic activity of normal and abnormal human liver with reference to anemia, 177
sickle cell, 721
sickle cell, in white American family, 1184
sickle cell, pathology of, 710
Splenic: See also Gaucher's disease
splenic, differentiated from splenomegaly, 1186
treatment, combined, 1451
Anencephaly: See Monsters
Anesthesia; pentobarbital sodium as preanesthetic agent, 464
Aneurysm, cerebral; case of congenital multiple arteriovenous intracerebral and extracerebral aneurysm with psychologic correlations, 1208
Angina, agranulocytic, 1194
agranulocytic, in diphtheria carrier, 429
agranulocytic; malign neutropenia caused by drugs, 189
agranulocytic, oral manifestations of, 456
agranulocytosis after aminopyrine, 1453
Diphtheritic: See Diphtheria
lymphatic, glandular fever and serum test of Paul and Bunnell, 446
Vincent's, postanginal sepsis; report of 9 cases, 913
Angiomatosis, Hemorrhagic Familial: See Telangiectasis
of retina, case of, 964
Angiosarcoma: See Sarcoma
Aniridia: See Iris, absence of
Anisocoria: See under Pupils
Ankylostoma; Ankylostomiasis: See Hookworm Infestation
Anorexia: See Appetite
Anthelmintics, action of copper on intestinal worms, 443
anthelmintic properties of certain alkyl phenols, 705
Anthocyaninuria: See Urine
Anthropometry: See also under Body; Growth; etc.
biometric data on normal Neapolitan newborn, 1432

Anti-Alpha Vaccine (Ferrán's): See Tuberculosis, vaccine therapy; Whooping Cough, therapy
Antigens and Antibodies; antígens in milk, 1158
circulating allergic antibody, 996
histamine in relation to intradermal reaction to allergens in sensitized persons, 997
ring precipitin test for estimating concentration of antibody in small amounts of immune serum, 1151
Antrum: See also Sinuses, nasal
maxillary, pathology of floor of, 1463
Anus, imperforate, case of, 1429
Aorta, coarctation, 158, 224
coarctation, importance and diagnosis, 680
coarctation in father and son, 157
coarctation, with infective endocarditis, 715
stenosis of inflammatory origin with differential study of acquired or congenital origin of bicuspid aortic valve, 704
Appendicitis, acute, 1226, *1296
acute, in epidemic parotitis, 917
acute, simulated by mesenteric lymphadenitis, 1227
acute, statistical survey of 8,000 cases, 469
and acute abdominal syndrome due to purpura, 1444
and acute inflammatory abdominal conditions in scarlet fever, 418
chronic, and congenital syphilis, 1467
chronic, anisocoria in, 1469
roentgenology in, 975
Schilling hemogram in, 1226
symptomatology and differential diagnosis, 974
Appendix, absence, congenital, 1429
absence in fetus with congenital syphilis, 410
foreign bodies in, report of case, 939
Appetite, Chinese method of acupuncture in treatment of nurslings with anorexia, 1423
loss of, and vomiting, 407
Apple diet in enteritis, 153
raw, diarrhea, 403, 1487
raw, in dysentery, 685
Arachnodactylia: See Fingers and Toes, abnormalities
Arginase: See Enzymes
Argyria: See under Silver
Arnold, D. P.: Dilatation and hypertrophy of heart, 479
Incidence and types of tuberculosis in children, 475
Noninfectious pneumonia of stillborn and new-born, 481
Aronson, J. D.: Effect of vaccination with BCG on infant and childhood types of tuberculosis with correlation of tuberculin reactions, roentgen diagnosis and mortality, 748
Arrhythmia: See also Heart, block; Heart, rate
auricular fibrillation as early toxic digitalis manifestation, 943
auricular flutter in new-born, 683
recovery from auricular fibrillation following diphtheria, 697
Arsphenamines, sensitization, patch tests in determination of, 710
Arteries: See also Aneurysm; Aorta; Blood, pressure; Pulse; Thrombosis; etc.
cerebral; arterial changes in brain in childhood, 1205
pulmonary, roentgen study by injection of contrast mediums, 1196
Arthritis: See also Gout; Rheumatism
acute polyarthritis with tubercle bacillemia, 1174
deformans; Still's disease cured by gold, 953
deformans; Still's disease with erythema multiforme, 953
deformity, 442
rheumatoid, treatment with gold salts, 1188
suppuration, caused by Bacillus suipestifer, 1156
tuberculous; articular foreign body in elbow, 1468
Arthrochalasis: See under Joints
Ascariasis, chenopodium oil in, 722
Asphyxia, intra-uterine pulmonary lesions associated with, 157
neonatorum; respiratory failure including so-called asphyxia neonatorum, 1431
neonatorum treatment by tracheal intubation, 683
neonatorum with pulmonary lesions, 157
Asthenia, climatic therapy in, 435
Asthma, bronchial, and states of acidosis and alkalosis in babies, 180
concentrated splenic extract in, 970
due to pollen; effect of low relative humidity at constant temperature, 942
pollen sensitiveness in children with, 947
primary tuberculosis, obstructive pulmonary emphysema and asthma with recovery, *118
reflex, pathology and treatment, 175
Astragalus, congenital fusion with scaphoid; bilateral; inherited, 680
Ataxia, hereditary; Friedreich's ataxia, 733, 1211
Atelectasis: See Lungs, collapse
Athletics, effect of alcohol on efficiency in, 1230
injuries in, manipulative surgery in, 1228
Athrepsia: See under Nutrition
Atmosphere: See Air
Atrophy: See also under names of organs and regions
Infantile: See under Celiac Disease
Muscular: See also Dystrophy, muscular
muscular, early infantile progressive spinal (Werdnig-Hoffmann), and its relation to myotonia congenita (Oppenheim), 1457
muscular; familial periodic paralysis and its transition into spinal muscular atrophy, 190
unilateral; surgical consideration of progressive hemiatrophy of face, 196
Atropine tolerance; negative action of serum of tolerant subjects, 742
Auricular Fibrillation: See Arrhythmia
Avitaminosis: See under Deficiency Diseases; and under names of deficiency diseases, as Beriberi; Scurvy; etc.

B C G: See under Tuberculosis
Babinski Reflex: See under Reflex
Bacilli: See Bacteria
Backward Children: See under Children
Bacteremia: See also under Diphtheria; Pneumococci; Septicemia; Streptococci; Tuberculosis; etc.
passage of bacteria from lungs into blood stream, 149
Bacteria: See also Diphtheria, bacilli; Pneumococci; Tubercle Bacilli; etc.
abortus and melitense group; brucellosis, 1433
Acidi-Lactici: See Bacteria, coli
air studies at higher altitudes, 148
Bordet-Gengou's: See Whooping Cough
Clostridium Welchii, endogenous infections, due to, 673
coli septicemia complicating pyonephrosis due to bilateral congenital ureteral strictures, 940
coli; urinary colibacillosis, 1169
colibacillus infections, 1434
Friedländer's: See Bacteria, mucosus capsulatus group
Gas: See Gangrene, gas
Lactobacillus acidophilus in production of dental caries, 478
Meningococci: See Meningococci
mucosus capsulatus group; symptomatology and clinical aspects of otitis caused by Friedländer's bacillus, 1464
Ohara's: See Dysentery
Pertussis: See Whooping Cough
Shiga's: See Dysentery
Sonne's: See under Dysentery
Streptococci: See Streptococci
suipestifer, as cause of suppurative arthritis, 1156
Balsam of Peru, hypersensitivity of skin to, 1225

Bang's Disease: See Undulant Fever
Banti's Disease: See Anemia, splenic
Barbital and Barbital Derivatives; cardiac irregularities produced by ephedrine and protective action of sodium barbital, 213
effect on embryo and on pregnancy, 742
nirvanol sickness, 194
pentobarbital sodium as preanesthetic agent, 464
toxicity; cisternal and lumbar drainage in coma from acute barbital poisoning, with observations on chronic barbital poisoning, 733
Barlow's Disease: See Scurvy
Barrier, Hemato-Encephalic: See Brain; Meninges, permeability
Basophils: See Erythrocytes, basophilic stippling
Behavior and developmental diagnosis, 1233
case of injury at birth; behavior aspects; neuropathology, 1234
disorders, medical approach to study, with analysis of 1,000 cases studied at child guidance home, 1205
study of 6 infants; diagnostic aspects; influence of thyroid on mental growth, 1236
Bender, L. F.: Pseudohypertrophic progressive muscular dystrophy; report on patients treated with amino-acetic acid, 745
Benjamin, E. L.: Enterogenous abnormalities; cysts and diverticula, *1126
Beriberi in nurslings, 1157
Biedl Syndrome: See Laurence-Biedl Syndrome
Bilirubin, in Urine: See Urine, bilirubin

BIOGRAPHIES:

Wadd, W.: Nursery mems and anecdotal biographies, *131

Birth control, dangers of chemical contraceptives, 978
Injuries: See under New-Born Infants
relation of month of birth to future of child, 1230
Black, R. A.: Enterogenous abnormalities; cysts and diverticula, *1126
Black, W. C.: Death from hemorrhagic encephalitis following treatment with sulpharsphenamine; report of 2 cases in syphilitic infants, *609
Quantitative Wassermann tests in diagnosis of congenital syphilis; clinical importance of Fildes' law, *1257
Bladder, calculi, large, 184
Blennorrhea: See Gonorrhea
Blepharophimosis: See under Eyelids
Bleyer, A.: Hair plucking, *336
Blindness, causes and prevention, 200
relation between photophobia and nyctalopia, 204
xerophthalmia and other frequently occurring diseases of eyes in relation to rate of blindness, 1216
Blister fluid, reagin content of, 172
Blood: See also Erythrocytes; Leukocytes; etc.
Acetone Bodies: See Blood, ketone bodies
alkali reserve and inorganic phosphorus content in healthy children and in children with various infections of respiratory tract, 1424
amino-acid in maternal blood and in that of umbilicus, 406
bactericidal power of, 435
bactericidal properties; avitaminosis and resistance to infectious diseases, 1158
calcium and inorganic phosphorus determination in 294 children, 898
calcium and phosphorus in premature infant, 1158
calcium and phosphorus, variations after administration of strontium salts, 150
calcium in new-born, 160
calcium, rôle of, 1423
Blood—Continued
carotene content; absorption of carotene, *273
cevitamic acid in; studies on metabolism of cevitamic acid in infantile scurvy, 1488
chemical values in children and adolescents aged from 8 to 16 years, 398
chlorides in meningitis, 192
cholesterol and creatine excretion in diagnosis and treatment of hypothyroidism, 1179
cholesterol content; variations in children with pulmonary tuberculosis after sojourn at high altitude, 168
cholesterol in cases of epileptic convulsions and of spasmophilia, 732
cholesterol in diabetes, 177
cholesterol of plasma in neonatal period, *84
coagulation; substances involved in coagulation of new-born; variations in fibrinogen content in normal infant, *99
coagulation; substances involved in new-born; prothrombin; quantitative and qualitative studies of platelets in normal infant, *590
coagulation; substances involved in new-born; studies of fibrinogen, *311
coagulation time, 1196
Diseases: See Anemia; Hematopoietic System; Leukemia; etc.
effect of posterior lobe of pituitary on, 1416
Electrolytes: See Electrolytes
fats and lipoids; exchange of lipids in umbilical circulation at birth, 900
fatty acids in; unsaturated fatty acid (vitamin F) deficiency, 1418
fibrinogen content in new-born, with regard to change in hemophilia neonatorum temporaria, 1163
fibrinogen; substances involved in coagulation of blood of new-born, *311
fibrinogen; substances involved in coagulation of blood of new-born; variations in fibrinogen content in normal infant, *99
groups and race, 1232
guanidine content, 905
influence of epinephrine on amount of water in, 1424
iron; values for hemoglobin determined by Newcomer method and compared with values calculated from iron content, 940
ketone bodies; periodic acetonemia, 720
lead in peripheral blood, 30 minute spectrographic micromethod for detection of, 146
macrocytosis in connection with catalase index, 399
nonprotein nitrogen constituents of blood and human milk, 1418
phosphatase, in normal and rachitic children, 1159
phosphorus; alkali reserve and inorganic phosphorus content of blood in healthy children and in children with various infections of respiratory tract, 1424
phosphorus and calcium content in premature infant, 1158
phosphorus and calcium determination in 294 children, 898
phosphorus; effect on blood content and on urinary elimination in experimental scurvy, 406
phosphorus; influence of thyroid and thymus on behavior of phosphatemia and glycemia, 406
phosphorus, necessary precautions to be taken in clinical determination, 399
physicochemical study of blood of 10 adults presenting Chvostek sign, 1455
Platelets: See also Purpura
platelets, after splenectomy, *765
platelets, quantitative and qualitative studies; substances involved in coagulation of blood of new-born; prothrombin, *590
pressure; mean dynamic arterial pressure, 1197
pressure of normal and tuberculous children, 435

Blood—Continued
sedimentation, rapid determination of, 1151
sedimentation rate in congenital heart disease, 410
sedimentation rate in rheumatic carditis, 713
sedimentation; simple method of estimating sedimentation rate of red blood cells, 149
sedimentation; value of sedimentation test in routine medical examinations and in certain diseases, 704
Serum: See Serum; and various subheads under Blood
studies of infantile sepsis, 947
Sugar: See also Dextrose, tolerance
sugar; chronic hypoglycemia, 676
sugar, epinephrine and regulation of dextrose, 1151
sugar estimation in 0.1 millilitre of blood by modified Folin-Malmros method, 397
sugar in mother's blood after fasting; its relation to birth weight of infants, *297
sugar; influence of thyroid and thymus on behavior of phosphatemia and glycemia, 406
sugar levels and dextrose tolerance in experimental poliomyelitis, *91
sugar; mental deterioration associated with convulsions and hypoglycemia; report of 2 cases, *575
sugar; relation between blood sugar and spinal fluid sugar, 151
sugar; relation between tissue growth, glycolysis and oxygen pressure, 398
sugar tolerance curves in toxic diphtheria, 920
sulfur compounds; glutathione content of blood of healthy children and nursing mothers, 1155
sulfur compounds; glutathione in blood in various diseases, 720
sulfur compounds; influence of certain hormones on glutathionemia in infancy, 397
transfusion, accidents attributed to, 181
transfusion, continuous drip with case records of large transfusions, 1229
transfusion in malnutrition, 1420
transfusion, simple apparatus for keeping citrated blood warm during injection, 942
Vessels: See Arteries; Capillaries; Endarteritis; Veins; etc.
vitamin A in, 155
volume, diminution in infantile intoxication, 907
Blum Phenomenon: See Chlorides, metabolism
Body: See also Height; Weight
build; relation of intracutaneous tuberculin reaction to physical constitution of Korean school children, 702
measurements; studies of school children in Slovakia, 474
standards of physical development for reference in clinical appraisement, 471
Bonar, B. E.: Blood in stools of new-born, *255
Bones: See also under names of bones
action of parathormone (Collip) on bone tissue, 1416
atrophy; Morquio's disease in 2 brothers, 485
atrophy; rare form of familial osseous dystrophy, 1443
calcium determination in vivo, 724
Deformities: See Abnormalities and Deformities; Poliomyelitis; Rickets; etc.
Diseases: See also Osteochondritis; Osteomyelitis; etc.; and various subheads under Bones
diseases, in relation to hyperparathyroidism, 707, 1181
Dystrophy: See Bones, atrophy
Fractures: See Fractures
fragilitas ossium tarda, 1187
growth; familial cleido-cranio dysostosis, 155
growth, indicated by circumference of wrist, 1231
growth; 2 cases of childhood myxedema, reported for purpose of emphasizing importance of bone-age studies, 941
Bones—Continued
injuries; manipulative surgery in athletic injuries, 1228
Marble: See Osteosclerosis
marrow; modified technic for sternal puncture and its value in hematologic diagnosis, 942
ossification praecox, 1452
Softening: See Osteomalacia
tuberculosis, diagnosis of osteo-articular tuberculosis, 1441
tuberculosis, pathologic anatomy based on phthisiogeneric theory of Ferrán, 436
Book Reviews:
Annuaro de eststistica demographo-sanitaria 1927-28, 232
Association pour le développement de l'hygiène maternelle et infantile, 232
Autoptisch kontrollierte Encephalogramme; F. Kruse and G. Schaetz, 492
Bacteriology of Milk; G. S. Wilson, 1492
Bacteriology of Typhoid, Salmonella and Dysentery Infections and Carrier States; L. C. Havens, 1492
Behavior Development in Infants; E. Dewey, 491
Blutungskrankheiten; H. Lehndorff, 492
Body Water; J. P. Peters, 998
Colwell's Daily Log for Physicians; J. B. Colwell, 232
Common Contagious Diseases; P. M. Stimson, 1493
Diabetic Primer for Children; E. M. Coleman and A. E. Fischer, 1493
Diagnosis and Treatment of Pulmonary Tuberculosis; J. B. Hawes 2d and M. J. Stone, 1250
Diphtérie maligne; R. Poinso, 758
Diseases of Endocrine Glands; H. Zondek, 231
Estudio radiologico de la neumonia en los niños; H. C. Bazzano, 758
Glands and Efficient Behavior; F. Mateer, 757
Growing Superior Children; I. N. Kugelmass, 756
Growth; Study of Johnny and Jimmy; M. B. McGraw, 231
Human Foot; D. J. Morton, 1249
Infant Nutrition; W. McKim Marriott, 232
Leitfaden der Kinderheilkunde; E. Stransky, 1250
Morbilidad y mortalidad en la edad preescolar; G. L. Bayley Bustamante, 1494
New Pathways for Children with Cerebral Palsy; G. G. Rogers and L. C. Thomas, 1249
Nourrisons débiles et prématuré; L. Ribadeau-Dumas and M. Lataste, 999
Otite latente del lattante in pediatria; R. Canino, 1494
Parathyroids in Health and in Disease; D. H. Shelling, 999
Patient and Weather; W. F. Petersen, 491, 1250
Pediatric Treatment; P. S. Potter, 232
Sífilis congénita en el niño; C. Torres Umaña, 1493
Speech in Childhood; G. Seth and D. Guthrie, 758
Stutterers; S. Blanton and M. G. Blanton, 1494
Surgeon General of Public Health Service of United States, Annual Report of, 1494
Your Child in Health and in Sickness; H. L. Dwyer, 1000
Bordet-Gengou Bacillus: See Whooping Cough, bacilli
Borovsky, M. P.: Raw apple treatment for diarrhea in pediatric practice, 1487
Bouillaud's Disease: See Rheumatic Fever
Boyd, E.: Weight of thymus and its component parts and number of Hassall corpuscles, *313
Boyd, G. L.: Atrophic pyelonephritis or nephritis secondary to congenital anomalies, 485
Bradshaw, P. S.: Use of adult serum as prophylactic measure in measles, 982

Brain: See also Cerebellum; Meninges; Nervous System; etc.
Aneurysm: See Aneurysm, cerebral
arterial changes in childhood, 1205
diseases; cerebral disturbances and their encephalographic diagnosis; pathologic encephalogram, 733
Encephalography: See Brain, roentgenography
hemangioma; associated facial and intracranial hemangiomas, *868
hemorrhage, intracranial, without paralysis, 451
hemorrhage, resulting from cisternal puncture, treatment of, 1207
hernia; cephalic monster with unusually large meningo-encephalocele, 1209
hernia; rhino-encephalocele, 725
influence of diencephalon and hypophysis on general autonomic function, 448
lesions, nervous mechanism of nystagmus and its significance in topical diagnosis of cerebral lesions, 198
neuroglia in formation of myelin in prenatal infantile brain, 190
phosphatide content in Niemann-Pick's disease and in infantile amaurotic idiocy, 735
residual psychic phenomena following traumatic lesions of head, 1458
roentgenography; apparatus for simultaneous displacement of spinal fluid and injection of air for encephalography, 1206
roentgenography in diagnosis of cerebral disturbances; pathologic encephalogram, 733
roentgenography in diagnosis of cerebral disturbances; performance of encephalography, 732
roentgenography; results of encephalography in epilepsy, 734
tuberculoma, associated with ear disease, 453
tumors; familial glioma (genetics of glioma), 194
wounds and injuries, after-effects of, 741
Breast Feeding: See Infant Feeding; Milk, human
histologic study of normal mamma in relation to tumor growth; early development to maturity, 950
Milk: See Milk, human
Bretonneau and first French literature on dothienenteritis, 928
Bright's Disease: See Nephritis
Brill's Disease: See Typhus
Bromide and Bromine, absence of deteriorating effects in epilepsy, 726
brominism, 473
permeability of meninges to, in course of diphtheria, 1434
permeability of meninges to, in healthy children, 732
Bronchi, Fistula: See Fistula
Foreign Bodies: See Foreign Bodies
roentgenography; bronchography, 1219
tuberculosis, 163
tuberculosis; obstructive pulmonary emphysema due to partial obstruction of bronchi by tuberculous lesions, *69
Bronchial Tree: See Bronchi
Bronchiectasis as source of scarlet fever dissemination, 161
atelectatic, roentgen-ray appearance of lung field in, 938
congenital; postmortem and pathologic investigation, 1191
lobectomy in one-stage, account of 48 cases, 469
Bronchitis, prognosis and treatment, 715
vena azygos lobe in child with, 176
Bronchography, 1219
Bronchopneumonia, acute, treatment of, 1197
classification of, with reference to bronchopneumonia in measles, 689
pathogenesis and prophylaxis in diphtheria, 690
prognosis and treatment, 715
Bronchopneumonia—Continued
subcutaneous generalized emphysema following bronchopneumonia and scarlet fever, 182
treatment, 719
treatment in infants under 1 year, 1444
unusual aspects of, 1446
Bronchoscopy: See Foreign Bodies
Brown, A.: Capillary resistance test as measurement of vitamin C deficiency, 478
Effect of mineral deficiencies on intestinal stasis, 477
Irradiated cholesterol in cure of rickets, 485
Brucella Abortus; Brucella Melitensis: See Bacteria, abortus and melitense group
Brucellosis: See under Bacteria, abortus and melitense group
Buhl's Disease: See Degeneration, fatty
Buphthalmos: See Hydrophthalmos
Burns, tannic acid in, 1227
Bursa, cystic hygroma of neck; 2 cases in Bulu children, *349
synovial, prenatal incidence, structure and development, 672
Butter, bovine tubercle bacilli in, 1177

Calcemia: See Blood, calcium
Calcification: See also Bones, growth; Parathyroid; Teeth
calcified abdominal glands, 980
calcinosis interstitialis progressiva, 188
of choroid plexus; report of case in child aged 4 years, *123
Calcinosis: See Calcification
Calcium and phosphorus studies; effect of prophylactic and curative doses of standardized viosterol on human tissues; necropsy report of 13 cases showing no tissue damage, 401
and phosphorus studies; effect of variation of calcium, phosphorus, and of vitamin D in diet on iron retention in rats, 401
Deposits: See Calcification
distribution of calcium and phosphorus in different organs and tissues of rats in deficiency and excess of parathyroid hormone, 151
effects on fetus of hypervitaminosis D and calcium and phosphorus deficiency during pregnancy, *302
excess of, as cause of rickets and osteomalacia in rabbits; histology of cranial bones, 1416
gluconate in eruptive fevers, 923
in Blood: See Blood, calcium
in human milk after administration of calcium, viosterol and parathyroid extract, 150
influence of local excess of calcium and phosphorus on healing of fractures, 464
metabolism in infantile tetany, *816
metabolism; rôle of mineral metabolism in incipient tuberculosis, 434
teeth of workers with cement, 474
tetany of new-born with deposit of calcium following parenteral administration, 219
Calvin, J. K.: Hematocolpos due to imperforate hymen, *832
Neuroblastoma in infant, 225
Cancer: See Sarcoma; Tumors; and under names of organs and regions
Canities: See under Hair
Capillaries of premature infant, 410
of skin of new-born, 415
resistance, studies of, 724
resistance test as measurement of vitamin C deficiency, 478
Carbohydrates: See also Dextrose; Glycogen; etc.
and development of experimental xerophthalmia, 405
Metabolism: See also Blood, sugar; Dextrose metabolism in juvenile diabetes, 988
Caries, Dental: See Teeth

Carotene: See also Vitamin A
absorption of, and decoloration following continued use, *249
metabolism, 150
parenteral use in treatment of vitamin A deficiency in albino rats, 676
Catalepsy, 727
Cataract, epilepsy and tetany, 193
parathyroid glands in relation to, 1214
sun-flower, in hepatolenticular degeneration (pseudosclerosis); report of case, 202
Celiac Disease, 944
case of Herter's disease with chronic pulmonary infection, 188
gastric secretion in, 1420
Gee's disease, 678, 1194
progress in early cases, 481
unusual features, 952, 954
Cells, physiology; new method for obtaining tissue fluid, 186
Cement, teeth of workers in, 474
Cerebellum, hemorrhage in boy of 14 years, 1460
Cerebrospinal Fever: See Meningitis
Cerebrospinal Fluid: See also Cisterna Magna, puncture; Spinal Puncture
analytic features in nonsyphilitic diseases of neuraxon, 958
cell count or globulin content not increased in case of tuberculous meningitis, 1176
changes in tetany, 1426
chlorides in meningitis, 192
dextrose content of lumbar and cisternal fluid, *17
in congenital syphilis, 1175
mumps complicated by meningitis with reference to, 696
sugar; relation between blood sugar and spinal fluid sugar, 151
Tubercle bacilli in: See under Tubercle Bacilli
Cerebrum: See Brain
Cesarean section, postmortem, 683
Chagas' Disease: See Trypanosomiasis
Character, development; cooperation with sciences of medicine and psychology in study of early infancy, 1454
Chauffard-Still's Disease: See Arthritis deformans
Chenopodium, in pediatrics, 1230
Chest: See Thorax
Chickenpox and herpes zoster, 429
and herpes zoster and encephalitis, 1171
and herpes zoster, relation between viruses of, 924
elementary bodies of zoster and their serologic relation to those of varicella, 428
fatal in a new-born infant, 920
influence on tuberculosis, 935
Child Welfare: See also Infant Welfare
adoption practices and the physician, 213
child guidance as medical function, 1470
child health activities in state department of health, 214
child health clinic, 1470
child hygiene in coal mining community, 472
health centers in rural public health service, 471
homeless boys' retreat, 213
institutional children and their development in first year, 217
medical science and "l'Hospice des enfants-assistés," 473
morbidity and mortality, statistics on nursing children of society for protection of minors of Barcelona, 977
movement in Sweden, 1230
nutrition work in Springfield, Ill., schools, 402
prenatal and postnatal care (2 puericultures), 978
public education in child care in France, 215
rural child health program and the pediatrician, 471
stations, attendance of infants at, 216
Children: See also Infants; New-Born Infants; Pediatrics
backward: relation of abnormal children in towns of Regensburg and Göttingen to social structure of entire population, 217
Growth: See Growth
imaginary companions, 447
Nervous and Mental Disabilities: See Nervous and Mental Disabilities; and under names of various diseases
psychoanalytic theory of play, 1210
relations of academic abilities to social classes and family size, 217
Chinese, children, growth in San Francisco, *1324
Chipman, S. S.: Prophylaxis of rickets in premature infants with vitamin D milk, *1
Viosterol in prophylaxis of rickets in premature infants, *594
Chlorides, hormone regulation of, 1156
in meningitis, 192
metabolism; Blum's phenomenon, 181
Chloropenia: See Blood, chlorides
Choked Disk: See Neuritis, optic
Cholecystitis: See under Gallbladder, inflammation
Cholelithiasis: See Gallbladder, calculi
Cholera Infantum: See Diarrhea
Cholesteremia: See Blood, cholesterol
Cholesterol in Blood: See Blood, cholesterol
irradiated, in rickets, 485
metabolism in essential xanthomatosis, *1372
Chondrodystrophy: See Achondroplasia
Chorea, acute toxic, case of, 1212
case of, 1459
etiology, clinical findings and treatment, 956
Huntington's, in twins, 1206
medicinal treatment of, 191
minor, hypnosis in, 195
nirvanol sickness in, clinical aspects, 194
treatment, 728
tubercle bacilli in blood and spinal fluid in, 183
Choriomeningitis: See under Meningitis
Choroid plexus, calcification; report of case in child aged 4 years, *123
Christian-Schüller Syndrome: See Schüller-Christian Disease
Chvostek sign, physicochemical study of blood of 10 adults presenting Chvostek sign, 1455
Cisterna Magna, puncture, technic and uses, 453
puncture, treatment of intracranial hemorrhage resulting from, 1207
Clawfoot: See Foot
Cleft Palate: See Palate
Climate in therapy of asthenic conditions in infancy, 435
Clostridium: See Bacteria, Clostridium
Coccyx, abnormalities; partial congenital absence of sacrum and coccyx and lower sacral roots, 408
Cod Liver Oil: See also Rickets
comparative antirachitic efficiency of irradiated ergosterol and cod liver oil for chickens and description of method for determination of rachitic weakness of legs of baby chickens, 154
prophylactic value of antirachitic agents, 5 year clinical study of, 677
Cohen, L. S.: See Solis-Cohen, L.
Cohn, D. J.: Comparative dextrose content of lumbar and cisternal cerebrospinal fluid, *17
Colds: See Respiratory Tract, diseases
Colitis, non-specific, in relation to deficiency disorders and anemia, 440
ulcerative, relation to bacillary dysentery, 173
Coloboma: See Eyelids, abnormalities; Macula Lutea

Colon: See also Gastro-Intestinal Tract; Intestines
absorption of dextrose, 400
dilatation, coprolith containing bismuth as complication of, 185
dilatation; Hirschsprung's disease treated by ganglionectomy, 1228
dilatation in infancy, 181
dilatation; peculiar case of Hirschsprung's disease, 1162
dilatation; report of case of Hirschsprung's disease, 709
dolichocolon, 180
Inflammation: See Colitis
roentgenography; relief pictures, 721
Stasis: See Intestines, stasis
Colostrum, diphtheria antitoxin in, 697
Communicable Diseases: See also Exanthem; Immunity; Measles; Meningitis; Syphilis; etc.
international action for control, 429
prevalence in United States, 425, 426, 1433
recent additions to knowledge; significance of, 919
relapses and recurrences, 687
secondary cases among nonimmune family contacts, 417
Concussion, Cerebral: See Brain, wounds and injuries
Conjunctiva, essential shrinkage of, in case of probable epidermolysis bullosa dystrophica, 1466
tuberculosis, and tuberculin allergy, 1215
yeast-like fungus infection of, 960
Conjunctivitis, Diphtheritic: See Diphtheria, conjunctival
of new-born; pathogenicity of inclusion virus for eyes of new-born and for female genital tract, 966
Phlyctenular: See Conjunctiva, tuberculosis
pseudomembranous, acute, with buccopharyngeal eruption and without diphtheria, 963
Constipation, prevention, 405
Constitution; importance of constitutional environment in training of children, 217
types; biotypology, 1230
Contagious Diseases: See Communicable Diseases
Contraception: See Birth Control
Convalescent Serum: See under Serum; and under names of various diseases
Convulsions: See also Epilepsy; Tetany; etc.
convulsive state, 732
in childhood, 189
mental deterioration associated with convulsions and hypoglycemia; report of 2 cases, *575
Cooley's Anemia: See Anemia
Cooperstock, M.: Carbohydrate metabolism in juvenile diabetes, 988
Copper, action on intestinal worms, 443
influence on hemoglobin formation, 483
removal from drinking water, 906
Coprolith: See under Feces
Cornea, familial degeneration in 6 generations: clinically observed in 13 members of last 3 generations, 1215
hematic disciform infiltration of, 964
opacities, local quinine therapy in, 1461
softening; xerosis in eye clinic, 1462
Corpus Striatum, congenital lesion of, as cause of "frog" child, 1211
Corpuscles, Hassall: See Thymus
Correction to article by G. B. Dorff, entitled "Maldevelopment and Maldescent of Testes" (50: 1429, 1935), 396
Coryza: See Respiratory Tract, diseases
Cowie, D. M.: Circulating allergic antibody, 996
Histamine in relation to intradermal reaction to allergens in sensitized persons, 997
Puncture biopsy as diagnostic measure in differentiation of solid tumors, 986
Coxa Vara: See Hip Joint, deformities
Crane, M. M.: Congenital thrombocytopenia, *1114
Substances involved in coagulation of blood of new-born; studies of fibrinogen, *311
Substances involved in coagulation of blood of new-born; variations in fibrinogen content in normal infant, *99
Craniotabes in 4 month old twins, 678
Cranium, Anomalies: See also Microcephaly; etc.
anomaly; Lückenschädel of new-born, 983
congenital malformations of skull and premature synosteosis of sutures, 1162
histology of cranial bones in experimental rickets and osteomalacia in rabbits, 1416
injuries, prevention in new-born, 414
Creatine and Creatinine in Urine: See under Urine
Creatinuria: See Urine, creatine
Cretinism: See also Myxedema
study of 6 infant cretins; diagnostic aspects; influence of thyroid on mental growth, 1236
Croup, infectious, acute, 457
infectious, acute, associated with specific diseases, 943
infectious, acute, nonspecific, 1184
Cryptorchidism: See Testicles
Cushing Syndrome: See Pituitary Body
Cysts: See under names of organs and regions, as Intestines, cysts; Lungs, cysts; Thymus, cyst; etc.

Danlos Syndrome: See Ehlers-Danlos syndrome
Dannenberg, A. M.: Effect of vaccination with BCG on infant and childhood types of tuberculosis, with correlation of tuberculin reactions, roentgen diagnosis and mortality, 748
Darrow, D. C.: Mental deterioration associated with convulsions and hypoglycemia; report of 2 cases, *575
Davidson, L. T.: Prophylaxis of rickets in premature infants with vitamin D milk, *1
Viosterol in prophylaxis of rickets in premature infants, *594
Davis, E. M.: Hydranencephaly (Cruveilhier), 227
Tuberous sclerosis, 227
Deaf-mutism, hereditary transmission of, 1219
Deafness: See also Deaf-Mutism; Ear; Hearing; Otitis Media
analysis of 4,000 cases studied during past 25 years, 207, 459
clinical observations, 460
methods of testing and management of deafened school child, 207
prevention, 458
vital function studies; analysis of group of children with impaired hearing, 457
Debility, Congenital: See Premature Infants
Defectives: See Feeblemindedness
Deficiency Diseases: See also Beriberi; Nutrition; Scurvy; Vitamins; and under names of various diseases
condition of endocrine glands of rats during avitaminosis, 152
nonspecific colitis in relation to deficiency disorders and anemia, 440
pathology of avitaminosis, 900
physiologic effects of vitamin deficiencies as summarized from recent experiments, 899
sympathetic ophthalmia provoked by ultraviolet radiation in avitaminosis, 904
Deformities: See Abnormalities and Deformities; and under names of organs and regions
Degeneration, fatty; cerebral type of Buhl's disease, 911
hyaline; hyaline bodies on disks, 1462
typus degenerativus amstelodamensis (de Lange), 1460
Dehydration, adequate hydration in infancy, 900
Hartmann's solution in, 214
Delinquency, juvenile, a product of the home, 742
juvenile, and psychiatry, 1205

Dementia Paralytica: See Paralysis, general
Deming, J.: Respiration in infancy; study of rate, volume and character of respiration in healthy infants during neonatal period, *823
Denenholz, E. J.: Letterer-Siwe's disease, 219
Letterer-Siwe's disease; splenohepatomegaly associated with widespread hyperplasia of nonlipoid-storing macrophages; discussion of so-called reticulo-endotheliosis, *499
Dentistry in relation to diseases of hematopoietic system, 707
primary and major indications for orthodontic treatment, 1481
Dentition: See under Teeth
Depression, Economic: See Economic conditions
Dermatitis exfoliativa; epidemic pemphigus or Ritter's disease of new-born, 684
exfoliativa, Ritter's disease after bullous impetigo in new-born, 1224
infectious febrile, 1223
Dermatochalasis: See under Skin
Dermatomyositis, 210
Dermatorrhexis: See under Skin
Devereux, R. T.: Liver extract in treatment of infection, 1472
Dextrose: See also Blood, sugar; Carbohydrates
absorption from colon, 400
and epinephrine, subcutaneous injection of, followed by gangrene, 1447
content of lumbar and cisternal spinal fluid, *17
metabolism, in hepatomegalies, 1194
Tolerance: See also Blood, sugar
tolerance and blood sugar levels in experimental poliomyelitis, *91
tolerance curve, hypoglycemic phase of, 400
Diabetes Insipidus: See also Schüller-Christian Disease
experimental; its relation to anterior and posterior lobes of hypophysis, 146
insipidus; xanthomatosis and syndrome of diabetic exophthalmic dysostosis, *353
Diabetes Mellitus: See also Blood, sugar
adrenal hemorrhage in normally delivered infant of diabetic mother, 683
blood cholesterol in, 177
coma, 703
growth of diabetic children, 937
heredity in, 172
hyperpyrexia in, 705
inheritance of, 1182
juvenile, carbohydrate metabolism in, 988
salivary secretion in, pathologic changes in, 724
the diabetic child, 712
Diaphragm, elevation, congenital, 159
Hernia: See Hernia, diaphragmatic
relaxation, 179, 1453
Diarrhea: See also Dysentery
apple diet in, 153, 403, 1487
blood transfusion in cholera infantum, 904
in infants, 902
physiopathologic changes and treatment, 679
raw fruits in treatment of, 679
therapy, new points of view, 1152
Diathermy: See also under names of diseases, as Poliomyelitis, treatment with diathermy; etc.
first impressions of short-wave therapy, 1187
Diathesis, Allergic: See Anaphylaxis and Allergy
exudative, ocular manifestation in, 1462
exudative; therapeutic effects of salt-free diets of nursing mothers on cutaneous exudative manifestations in nurslings, 406
hemorrhagic, arrest of severe hemorrhages in all forms of, and in hemophilia by parenteral administration of vitamin C, 152
hemorrhagic, more especially essential thrombopenia, treatment with vitamin C, 1460
hemorrhagic; prognosis and management of bleeding tendency, 721
hemorrhagic, sporadic, 188
Dick Test: See under Scarlet Fever
Diet: See also Food; Infant Feeding; Nutrition; Vitamins; and under names of various diseases and foods
basal metabolism in relation to dietary intake, *1039
effect on creatinuria; normal variations; creatine tolerance tests and effect of aminoacetic acid in normal infants, *30
ketogenic; and ketosis; application to treatment of epilepsy and infections of urinary tract, 1207
Digestion: See Stomach, physiology
Digitalis, toxicity, auricular fibrillation as early manifestation, 943
Diphtheria, allergy and focal reactions in, 692
antiserum followed by acute articular rheumatism, 921
antiserum; is Ramon's flocculation specific reaction between toxin and antitoxin? 700
antiserum, permeability of meninges of healthy children to, 732
antiserum; practicability of Ramon's flocculation reaction, 700
antiserum; speed of Ramon's flocculation, 926
antiserum; studies on Ramon's flocculation; influence on flocculation of physicochemical factors, 699
antitoxin in blood of population in a town of Java, 696
antitoxin in human colostrum and in mother's milk, 697
antitoxin, specific effect on animals, 697
auricular fibrillation following recovery from, 697
bacilli, antigenic relation of gravis strains, as compared with Park 8 strain, 915
bacilli; attenuation and toxin production in fusion-free peptone mediums; in synthetic mediums, 421
bronchopneumonia in pathogenesis and prophylaxis of, 690
carrier; does presence of diphtheria bacilli in nose and auditory meatus involve danger of infection for environment of bacillus carrier? 695
carrier, with agranulocytic angina, 429
carriers and vaccination, 691
carriers, newer management of, 1166
conjunctival, catarrhal, 917
cutaneous, 916
cutaneous, in congenital syphilis; report of case, 417
cutaneous, epidemiologic significance of "atypical" diphtheria of skin, 695
cutaneous reaction to diphtheritic toxin and antitoxin content of blood, 689
extra-faucial, with palatal paralysis, 1166
immunization, 424, 430, 917
immunization by means of single dose of alum-precipitated toxoid, 685
immunization, combined active and passive, 688
immunization in prevention of specific fevers, 429
immunization in private practice, 913
immunization in school, 424, 429
immunization; results obtained in relatively small community using single dose toxoid method, 916
immunization with single injection of highly purified formol toxoid and Al $(OH)_3$, 915
immunization with toxoid and injections of antitoxin, 688
in new-born, 921
in tropics; subclinical disease in Negroes, 918
in Wisconsin, 427
interference in Schick test by diphtheria antitoxin injection, 429
is prognosis becoming graver? 1169
laryngeal, imitated by necrosis in scarlet fever, 925
"malignant;" are there anaerobic strains of diphtheria in "malignant diphtheria"? 695
malignant, diagnosis and treatment, 918

Diphtheria—Continued
malignant, serum therapy in, 922
myocarditis in, 423
Paralysis: See under Paralysis
permeability of meninges to bromide and to diphtheria antitoxin in course of, 1434
prophylaxis by combined immunization, 695
prophylaxis; evaluation of modern methods, 430
prophylaxis, recent trends in, 919
pseudotabes following, 693
renal function in, 923
Schick test and active immunization in relation to epidemic diphtheria, 920
Schick test, antitoxin titer and susceptibility to diphtheria, 925
Schick tests in Nigerian natives, 685
serum, efficacy, 924
serum, efficacy in experiments on animals, 694
serum therapy; relation between hematologic and clinical findings, 1434
toxic, therapy; significance of sugar-tolerance curves and value of insulin, 920
toxin; experiments with concentrated diphtheria toxin digested with formaldehyde, 924
toxin; persistence of diphtherotoxin in subcutaneous tissue of guinea-pigs, 1434
toxin; precipitation of diphtheria anatoxin by alum, 927
toxin, purification by calcium phosphate, 699
toxin, rapid method of hyperimmunization of horses against, 699
toxoid, alum-precipitated, 419
toxoid; experience with alum-precipitated toxoid in Virginia and observations on reaction, 416
toxoid, preparation of, absorbed on calcium phosphate, 914
treatment, 1434
treatment; should antitoxin treatment be complemented by nonspecific antigen therapy? 692
treatment, with antitoxic serum, new method, 916
vaccination, nasal, 162
vulvovaginal, 694
water metabolism in, (reaction of McClure and Aldrich), 162
Directory of pediatric societies, 233, 493, 759, 1001, 1251, 1495
Disease, seasonal curves, 977
Dislocations: See under names of joints
Diverticulum, Meckel's: See Intestines, diverticula
Dolan, D. J.: Heredofamilial cleft foot, *338
Dolichocolon: See under Colon
Dothienenteritis: See Typhoid
Drake, T. G. H.: Irradiated cholesterol in cure of rickets, 485
Drugs; pharmacology and pharmacodynamics of childhood, 216
use and abuse in treatment, 1471
Ductus Arteriosus, tetralogy of Fallot with right aortic arch and redundant left ductus arteriosus, *1256
Duke-Filatov's Disease: See Fourth Disease
Duncan, J. E.: Method of preserving breast milk; study of its clinical application, *249
Duodenum, Ulcer: See also Peptic Ulcer
ulcer, 2 cases, 706
ulcers, melena vera neonatorum due to, 1165
Dwarfism: See also Achondroplasia
endocrine, 171; correction, 666
in cases of heredofamilial disease of central nervous system, possible explanation, 1206
in pediatrics, 182
renal, and glycosuria, 955
renal, associated with valvular obstruction of posterior urethra, 951
renal rickets, 1203, 1451
Dyke, C. G.: Xanthomatosis and syndrome of diabetic exophthalmic dysostosis, *353
Dyschondroplasia: See Achondroplasia
Dysentery, 1171. See also Diarrhea
Amebic: See Amebiasis; Liver, abscess, amebic
apple diet in, 685
bacillary, bacteriophage in diagnosis of, 421
bacillary, clinical and bacteriologic study of 35 cases, 1165
bacillary, in Rockhampton with account of distribution and type in other states, 1167
bacillary; relation to ulcerative colitis, 173
bacillary; sodium thiocyanate in prophylaxis and treatment, with special emphasis on Shiga type, 424
bacilli, vulvovaginitis due to, 693
cardiac disturbances in, pathology and therapy, 692
epidemic of 1933 in Budapest, 693
in Denmark, 163
outbreak caused by Sonne type bacillus, 916
specific prophylaxis and therapy in summer diarrhea, 907
treatment with extract of posterior hypophyseal lobe, 924
Dysostosis: See under Bones, growth; Schüller-Christian Disease
Dyspituitarism: See under Pituitary Body
Dystrophy: See also Foot, dystrophy; Hand, dystrophy; etc.
acid-base equilibrium in dystrophic infants, 152
alimentary, stages of curability and incurability in, 1423
Muscular: See also Myotonia
muscular; glycine in, 177
muscular; myelomuscular dysplasia, 193
muscular, pseudohypertrophic, 727, 728
muscular, pseudohypertrophic progressive; preliminary report on patients treated with amino-acetic acid, 745
muscular, treatment with gelatin, 708
muscular treatment with glycine, 1449
Osseous: See Bones, atrophy
pellagra and "unapparent dystrophy," 1424

Ear: See also Deafness; Mastoiditis; Otitis Media; etc.
dyschondrogenesia, 968
examination; rôle of otoscope in otology in infants, 463
infections, acute; method of determining etiologic agent in, 1220
infections, concentrated streptococcus serum of New York State Department of Health in, 456
internal; labyrinthitis and sinus thrombosis complicating suppuration of middle ear, 206
middle, pressure and auditory acuity, 455
pathologic changes in late congenital syphilis, 1174
rupture of suppurative cervical glands into external auditory canal, 205, 1217
Echinococcosis of lung, 2 cases, 441
Economic conditions; further studies on growth and economic depression, 677
Ectopia Cordis: See Heart, displacement
Eczema, 970, 1225, 1466. See also Diathesis, exudative
concentrated splenic extract in, 970
device for control of hands in, 976
infantile, 1222
infantile, routine treatment, 970
sensitization in, 1222
treatment in childhood, 212
urinary proteose in, 210
vitamin D in treatment of, 1217
vitamins in treatment of, 1463
Edema, absorption; influence of ultraviolet rays on time of absorption and on activity of edema-producing substances, 398
angioneurotic; critical review of 170 cases of urticaria and angioneurotic edema followed for period of from 2 to 10 years, 211
cardiac; acute cardiac pulmonary edema and its reflex mechanism, 185, 443
McClure and Aldrich reaction; water metabolism in diphtheria, 162
Pulmonary: See Lungs, edema

Education, cooperation with sciences of medicine and psychology in study of early infancy, 1454
Egg yolk, vitamin D content, 402
Ehlers-Danlos syndrome; dermatorrhexis with dermatochalasis and arthrochalasis, *1403
Elbow, articular foreign body in, 1468
malformation, congenital, 408
Electrocardiogram: See under Heart
Electrolytes, changes in distribution of body water accompanying increase and decrease in extracellular electrolyte, 1419
Elghammer, H. W.: Spontaneous tension pneumothorax in 4 year old girl, 753
Embolism, pulmonary, experimental, associated with venoclysis, 898
Embryoma, abdominal tumors; report of series of cases, 993
Emesis: See Vomiting
Emphysema, pulmonary, obstructive, due to partial obstruction of bronchi by tuberculous lesions, *69
pulmonary: primary tuberculosis, obstructive pulmonary emphysema and asthma with recovery, *118
subcutaneous, complicating pneumonia, 954
subcutaneous, generalized, following bronchopneumonia and scarlet fever, 182
Empyema, acute, 463
analysis of 103 cases, 468
autopyotherapy, 973
diagnosis and treatment, 740
incidence of various types of pneumococci in infections other than pneumonia, 948
opsonic index in infants with, 179
rib resection with open drainage vs. non-open method, 468
staphylococcic, and pyopneumothorax, 1227
Encephalitis, acute (St. Louis type), pathologic changes in, 450
anatomicoclinical study of pallidal syndrome in, 736
complicating measles, 982
epidemic, of St. Louis type, 726
epidemic, recent advance in treatment, 728
epidemic (St. Louis), clinical features of, 448
etiology of 1933 epidemic, 449
following German measles, 220
hemorrhagic, death from, following treatment with sulpharsphenamine; report of 2 cases in syphilitic infants, *609
herpes zoster and chickenpox, 1171
in children, 193
infectious, 1211
postencephalitic personality disorders, 446
postvaccinal, in Netherland Indies, 1171
problem, 726
rare psychic and neurologic sequels in 2 cases following measles, 688
symptoms and diagnosis, 449
treatment, new, 1454
treatment of postencephalitic disturbance with large doses of atropine, 455
Encephalocele: See Brain, hernia
Encephalography: See Brain, roentgenography
Encephalomeningitis: See Meningo-Encephalitis
Encephalomyelitis disseminata following ascending neuritis, 1207
simulating diphtheritic paralysis, 1211
subacute, during course of scarlet fever, 922
Endarteritis: See also under Arteries
coronary, in acute rheumatism, 713
Endocarditis, infective, coarctation of aorta with, 715
malignant, following gonococcic abscess of myocardium, 1447
Rheumatic: See under Rheumatic Fever
Endocrine Glands: See also Endocrine Therapy; Hormones; Metabolism; and under names of glands
and anatomic and pathogenic considerations of myopathies, 1455
and dwarfism, 171; correction, 666
and fever production, 179
disorders of internal gland secretion, 945
of rats, condition during avitaminosis, 152
studies, 1185
Endocrine Therapy, clinical results, 486
results in mongolian idiots, 448
status of endocrine research, 984
Endothelioma: hemangio-endothelioma of temporal bone, 438
Energy Exchange: See Metabolism
Engelfried, J.: Circulating allergic antibody, 996
Histamine in relation to intradermal reaction to allergens in sensitized persons, 997
Enteric Fever: See Typhoid
Enteritis: See Colitis; Diarrhea; Dysentery; etc.
Enuresis: See Urination, incontinence
Enzymes, hepatic and renal arginase with reference to age variations, 1159
Eosinophilia in allergic conditions, 1451
Eosinophils: See also Leukocytes
after operations, 975
problem, 723
Ephedrine, cardiac irregularities produced by, and protective action of sodium barbital, 213
Epidermolysis bullosa dystrophica, essential shrinkage of conjunctiva in probable case of, 1466
bullosa hereditaria letalis, (congenital nonsyphilitic pemphigus); review with description of new disease, 1164
Epiglottis, cyst, congenital glosso-epiglottic, 967
Epilepsy: See also Sclerosis, tuberous
blood cholesterol in cases of epileptic convulsions and of spasmophilia, 732
bromides in, absence of deteriorating effects, 726
encephalography in, results, 734
in 3 pairs of identical twins, 1208
pediatric point of view, 729
tetany, cataract and epilepsy, 193
treatment; ketosis and ketogenic diet, 1207
Epinephrine: See Suprarenal Preparations
Equinovarus: See under Foot
Erb, I. H.: Complications of Meckel's diverticulum, 476
Ergosterol, Irradiated, antirachitic efficacy of irradiated cholesterol, 1418
calcium and phosphorus studies; effect of prophylactic and curative doses of standardized viosterol on human tissues; necropsy report of 13 cases showing no tissue damage, 401
comparative antirachitic efficiency of irradiated ergosterol and cod liver oil and description of more reliable method for determination of rachitic weakness of legs of baby chickens, 154
effect of generous but unfortified diet on rachitic rats, 991
effect of moderately large doses on tuberculous children, 163
effect of parenteral administration to rachitic infant, 1156
in prophylaxis of rickets in premature infants, *594
prophylactic value of antirachitic agents, 5-year clinical study of, 677
Eruptions: See Exanthems; Skin, diseases; etc.
Erysipelas, generalized, in 2 month old infant; multiple residual abscesses; pharyngolaryngeal abscess of pyriform sinus opened surgically by cervical route; recovery, 687
in young children, 928
purulent complications in new-born, 684
treatment with ultraviolet light, 428
Erythema annulare rheumaticum, 228
infectious, 691
infectious, or rubeola? 1171
multiforme in Still's disease, 953
nodosum and focal infection, 1466
nodosum and scarlet fever, 692
nodosum and tuberculosis, 1442
nodosum associated with negative reaction to tuberculin, 1440

Erythema—Continued
nodosum in subjects with preexisting tuberculosis, 1176
nodosum, local blood picture in, 1224
nodosum, 120 cases of, 1225
nodosum, pathogenesis of, 1224
Erythredema: See Acrodynia
Erythroblastosis: See Hematopoietic System
Erythrocytes, agglutinins for sheep and rabbit erythrocytes in human sera, 673
basophilic stippling in, in healthy infants in Mukden, 672
diameter in acholuric family jaundice and effects of splenectomy, 951
in acidosis and alkalosis, 1420
in icterus gravis neonatorum, *104
macrocytosis in connection with catalase index, 399
mechanism of increased fragility in congenital hemolytic jaundice, 703
Sedimentation: See Blood, sedimentation
staining of processes (flagella) of human erythrocytes, 148
values for red blood cells of average infants and children, *775
Erythrodermia, maculopapular; pityriasis lichenoides et varioliformis acuta (Habermann), 1221
Escherichia: See Bacteria, coli
Esophagus, achalasia, 953
atresia, congenital, 408
atresia, congenital, with tracheobronchial fistula, *1119
diaphragmatic hernia at esophageal hiatus, short esophagus and thoracic stomach, 680
malformation, rare, congenital, 908
obstruction, congenital, 159
obstruction, congenital, specimens from case of, 1429
perforation, spontaneous, case of, 442
stricture, congenital, 681
stricture, following corrosive burns, prevention of, 178
Ethisms, psychic, 1457
Ethyl Alcohol: See Alcohol
Eugenics, what it is and what it is not, 470
Exanthem; calcium gluconate in eruptive fevers, 923
Subitum: See Roseola
Exomphalos: See Hernia, umbilical
Exostoses, in unusual case of Madelung's disease, 1193
Extremities, abnormalities; "frog" child; congenital lesion of corpus striatum? 1211
paralysis; acrodynia with quadriplegia, 211
Exudates, rheumatic; cytologic studies on rheumatic fever, 673
Exudative diathesis, advantages of pure milk in treatment of, 905
Eyelids, abnormalities; so-called coloboma of macula lutea, 1463
abnormalities; tooth in lid and palpebral coloboma, 201
blepharophimosis, congenital, uncomplicated heredity of, 966
surgery; xerophthalmia and its treatment; new indication for central tarsorrhaphy, 962
Eyes: See also Orbit; Vision; and under names of special structures and diseases of eyes
Abnormalities: See also under special structures of the eye
abnormalities; heredity of congenital uncomplicated blepharophimosis, 966
abnormalities; microphakia and spherophakia with glaucoma, 959
abnormalities; papilla nervi optici primitiva epithelialis, persistence of, 1217
abnormalities; rare form of persistent pupillary membrane with microphthalmos, 201
diseases; ocular manifestation in exudative diathesis, 1462
gonococcus infection, 422
headaches in relation to ocular conditions, 201
hyaline bodies on disks, 1462
Eyes—Continued
melanosis, benign congenital, 962
papillary changes of nasal origin, 202
parasites; internal anterior ophthalmomyiasis; report of case, 1214
parasites; ophthalmomyiasis subretinalis, 1460
sarcoma, primary, of inferior oblique muscle, 963
tumor; persistence of papilla nervi optici primitiva epithelialis, 1217

Faber, H. K.: Quantitative Wassermann tests in diagnosis of congenital syphilis; clinical importance of Fildes' laws, *1257
Face, deformities due to perennial nasal allergy, 458
hemangioma, associated with intracranial hemangioma, *868
hemiatrophy (progressive), surgical consideration, 196
tumor, case for diagnosis, 954
Fallopian tubes; hysterosalpingography in newborn, 415
Fallot's Tetralogy: See Heart, abnormalities
Farmer, C. J.: Metabolism of cevitamic acid in infantile scurvy, 1488
Fat, abnormal storage, 1448
as genetic factor in nutritional disturbances of nursling, 906
fatty degeneration; cerebral type of Buhl's disease, 911
metabolism of adolescent girls, *1083
metabolism, spinal mechanisms of, 199
necrosis; cytosteatonecrosis in new-born, 1162
necrosis; subcutaneous adiponecrosis in newborn, 909
sparing action on vitamin B; influence of levels of protein and vitamin G, 1419
sparing action on vitamin G, 1419
Faust, O. A.: Primary tuberculosis, obstructive pulmonary emphysema and asthma with recovery, *118
Feces, Bacteria: See Intestines, parasites
blood in stools of new-born, *255
coprolith containing bismuth as complication of Hirschsprung's disease, 185
relationship between diseases of pancreas and hydrolysis of carbohydrates in feces, 153
Fecundity; relations of academic abilities to social classes and family size, 217
Feeblemindedness: See also Idiocy; Laurence-Biedl Syndrome; etc.
heredity and mental deficiency, 1211
physical and mental training in mental deficiency due to birth lesions, 956
typus degenerativus amstelodamensis (de Lange), 1460
Feet: See Foot
Femur, angiosarcoma, in new-born, 224
fracture, statistical analysis of 285 cases, 470
osteochondrosis of, 719
Fenton, M.: Circulating allergic antibody, 996
Ferry Antitoxin: See Meningitis, meningococcic
Fetus, congenital absence of appendix with congenital syphilis, 410
effects on fetus of hypervitaminosis D and calcium and phosphorus deficiency during pregnancy, *302
extrasystoles observed in utero, 682
glycogen in, postmortem disappearance of, 1432
heart beat, changes in rate in response to vibratory stimuli, *583
heart muscle of human fetus, histologic investigation, 910
intra-uterine fracture of leg, 1160
kidney, histology of, 412
Pathology: See also Achondroplasia; Hydrocephalus; etc.
Fever: See also Temperature, Body; etc.
Alimentary: See Nutrition, disturbances
immunization in prevention of specific fevers, 429
pathothermia, 1459
pericoronitis of first molars as cause of, 1445
production; endocrinosympathetic factor in, 179

Fibrinogen in Blood: See Blood, fibrinogen
Fibroleiomyoma, of left pulmonary vein, 1196
Fibroma; fibro-adipose tumor causing congenital hypertrophy of foot, 1161
Fibula, absence of, congenital, 1429
Fifth Disease: See Erythema, infectious
Figs, vitamin content of, 901
Filatov-Duke's Disease: See Fourth Disease
Fildes' law, clinical importance of, *1257
Fingers and Toes, abnormalities; arachnodactylia, 225
abnormalities; arachnodactylia or Marfan's syndrome; familial and hereditary ocular defects, 1192
abnormalities; bilateral anomaly of big toes, 410
abnormalities; case of dystrophia mesodermalis congenita (Marfan) or arachnodactylia, 412
abnormalities; dystrophies and malformations of fingers in hereditary syphilis, 435
abnormalities; hereditary syndactylism and polydactylism, 408
Fingersucking, device for control of hands, 976
Fisher, W. E.: Intrapericardial teratoma, *1397
Fistula, lacrimal, congenital, 965
recto-urinary, congenital in girl, 409
tracheobronchial, with congenital atresia of esophagus, *1119
Flatfoot: See Foot, flatfeet
Folliculitis, pustular, superficial, of new-born, 413
Food: See also Diet; Nutrition; Vitamins; and under various foods, as Milk; Soy beans; etc.
allergy, leukopenic index in, 1183
and dental diseases, 977
"digestibility" as determined by radiography, 1421
poisoning, ingestion of staphylococcus exotoxin by human volunteers with special reference to staphylococcic food poisoning, 676
poisoning, is special variety of staphylococcus concerned in? 149
Foot, clawfoot, etiology and treatment of, 1226
cleft, heredofamilial, *338
dystrophy, associated with familial bilateral macular coloboma, 961
flatfeet, 739
hypertrophy, congenital, 1161
lines in hands and feet, 1232
paralyzed; surgical orthopedic treatment of, 975
talipes equinovarus, 739
tendon reflexes of, 1209
Foreign Bodies: See also under names of organs and regions, as Appendix, foreign bodies in; Vagina, foreign bodies in
bronchial, extraction of metallic foreign body from right bronchus under roentgen control, 178
bronchial foreign bodies (nuts), 710
laryngeal; unusual laryngeal foreign bodies, 460
removal from lower air passages with aid of Haslinger bronchoscope, 210
trachea, bloodless removal of foreign bodies from trachea, 208
Fourth Disease, or Filatov-Dukes' disease, and like eruptions, 926
Fractures: See also under names of bones
healing, influence of local excess of calcium and phosphorus on, 464
intra-uterine, of leg, 1160
timely course of healing processes in, 975
unusual feature of celiac disease, 954
use of skeletal traction; presentation of new use, 1469
Frambesia, fatality from exacerbation of latent tuberculosis due to thiobismol in case of yaws, 1437
Frenulum labii, rational treatment for abnormal attachment, 1477
Friedländer Bacillus: See Bacteria, mucosus capsulatus group
Friedman, E.: Tuberculosis of adrenal glands in child of 12 with necropsy, *113
Friedman, I.: Changes in lung volume during treatment with artificial pneumothorax for lobar pneumonia; report of 3 cases, *523
Friedreich's Ataxia: See Ataxia, hereditary
"Frog" Child: See Extremities, abnormalities
Froin Syndrome: See Xanthochromia
Frontal Sinus, development and lack of development of accessory nasal cavities, 463
Fruits, raw, in diarrheas, 679
Fungi: See Actinomycosis; Mycosis; Ringworm; etc.
Furunculosis, treatment, 1467

Gallbladder, calculi and cholecystitis, 465
inflammation, acute, with gallstone in child, 465
inflammation and calculi, 465
strangulation, report of case with perforation and gangrene in boy of 17, 467
Ganglioneuroma in neck of child, 938
Gangrene following subcutaneous injection of dextrose and epinephrine, 1447
gas; endogenous infections by Clostridium Welchii, 673
of extremity in new-born, 682
Gastric Juice: See Stomach, secretion
Gastro-Intestinal Tract: See also Colon; Intestines; Stomach; etc.
diseases, blood transfusion for, 1420
disease; iron-deficiency anemia; its association with gastro-intestinal disease, achlorhydria and hemorrhage, 948
Gaucher's Disease, chemical examination of Gaucher's spleen, 445
fifth patient in one family, 179
splenectomy in, 185, 1468
Gee's Disease: See Celiac Disease
Genitals: See also Genito-Urinary Tract; Urinary Tract; and under names of genitals, as Testicles; Vagina; etc.
diseases; pathogenicity of inclusion virus for eye of new-born and for female genital tract, 966
physiology in new-born, 1163
Welander's ulcer, 1452
Genito-Urinary Tract: See also Genitals; Testicles; Vulva; etc.
anomalies, atrophic pyelonephritis or nephritis secondary to, 485
foreign bodies in, 227
pus in; diagnosis and treatment of infection of urogenital tract, 941
Genther, I.: Metabolism of adolescent girls; excretion of creatinine and creatine, *1268
Germ cells, disturbances in developmental functions; case of anencephaly, 1460
Gerson Diet: See Tuberculosis
Gesell, A.: Case of injury at birth; behavior aspects, 1234
Comparative study of 6 infant cretins under treatment; influence of thyroid on mental growth (illustrated by cinema), 1236
Developmental diagnosis of infant behavior, 1233
von Gierke's Disease: See Glycogen, in liver and muscle
Gigantism, specimens from case of congenital hyperplasia of suprarenal cortex, 1429
with splanchnomegaly, 440
Gittleman, I. F.: Infantile tetany; metabolic study, *816
Glands: See Lymph Nodes; and names of individual glands
Glandular Fever: See also Mononucleosis, infectious
lymphatic angina and serum test of Paul and Bunnell, 446
Glaucoma: See also Hydrophthalmos
infantile, 959
microphakia and spherophakia with, 959

Glioma, familial, genetics of, 194
of retina, report of case with intracranial extension, 199
Globus Pallidus: See under Lenticular Nucleus
Glucose: See Dextrose
Glutathione in Blood: See Blood, sulphur compounds
Glycemia: See Blood, sugar
Glycine: See Acid, amino-acetic
Glycogen, in liver and muscle; enlargement of heart due to abnormal glycogen storage in von Gierke's disease, 704
in liver and muscle; von Gierke's glycogen accumulation disease, 176
in liver and muscle; hepatomegaly; von Gierke's disease? 952
in liver and muscle; storage diseases, 1448
in liver and muscle; thesaurismosis glycogenica (von Gierke), 705
postmortem disappearance in new-born and in fetus, 1432
Glycolysis: See Blood, sugar
Glycosuria: See Urine, sugar
Goiter: See also Thyroid
congenital, 721
exophthalmic; Graves' disease in child, 952
iodine tolerance of children with, 1199
iodized salt in prophylaxis; review of literature on use of, 985
Gold, H.: Studies on prophylactic immunization against whooping cough, 1473
Gold in treatment of disease, 1470
salts, in rheumatoid arthritis, 1188
Goldbloom, A.: Shakespeare and pediatrics, *653
Gonococci: See also Gonorrhea
abscess of myocardium with malignant endocarditis, 1447
dissociation, 148
Ophthalmia, Due to: See Ophthalmia
Gonorrhea: See also Gonococci
lyzed gonococci in treatment of cervicovaginitis, 440
ocular complications of, 422
therapy, 443
vaginitis due to, amniotin in, 171
vaginitis due to, diathermy in, 944
vaginitis due to, new treatment, 1166
vaginitis due to, pyridium in, 439
vulvovaginitis due to, etiology of, 1170
Gordon Test: See Lymphogranuloma, diagnosis
Gout and aleukemic leukemia in boy aged 5, 713
Grand, M. J. H.: Heredofamilial cleft foot, *338
Granulocytopenia: See Angina, agranulocytic
Granulopenia: See Angina, agranulocytic
Grapes, vitamin content of sultanina (Thompson seedless) grapes and raisins, 901
Graves' Disease: See Goiter, exophthalmic
Greenberg, M.: Congenital cardiac anomalies in infants, report of 5 cases, *1356
Greene, D.: Recurrence of pneumonia in infancy and in early childhood, with special reference to prognosis, *284
Greengard, J.: Malignant thymoma, 222
Greenwald, H. M.: Associated facial and intracranial hemangiomas, *868
Grip: See Influenza
Grisamore, T. L.: Primary and major indications for orthodontic treatment, 1481
Growth, adolescent, in relation to basal metabolism, *1014
age changes in physical resemblance of siblings, 742
and sparing action of fat on vitamin B; influence of levels of protein and vitamin G, 1419
and sparing action of fat on vitamin G, 1419
effects of lactose on, 1419
further studies on growth and economic depression, 677
increased in school children since war, 1229
of diabetic children, 937
of oriental children in San Francisco; a contrast, *1324
specific effect of vitamin A on, 403
Guanidine, experimental research on relation of potassium and guanidine; pathogenesis of spasmophilia, 731
in Blood: See Blood, guanidine
intoxication, acidosis of, 675
Gums, inflammation: pericoronitis of first molars as cause of fever, 1445

Habit; psychic "ethisms," 1457
Hair plucking, *336
premature grayness, 1222
Hand, deformities; unusual case of Madelung's disease, 1193
dystrophy, associated with familial bilateral macular coloboma, 961
lines in hands and feet, 1232
Hand's Disease: See Schüller-Christian Disease
Hand-Schüller-Christian Disease: See Schüller-Christian Disease
Hanganatziu-Deicher Reaction: See Agglutination, hemagglutination
Hanner, J. P.: Respiration in infancy; study of rate, volume and character of respiration in healthy infants during neonatal period, *823
Harelip, and cleft palate; sequence of operations for surgical correction, 1478
histologic study of anatomic structures forming oral cavity, 672
primary and major indications for orthodontic treatment, 1481
Harley, L. M.: Abdominal tumors in children; report of series of cases, 993
Harper, P.: Changes in lung volume during treatment with pneumothorax for lobar pneumonia; report of 3 cases, *523
Treatment of lobar pneumonia in children by artificial pneumothorax, *536
Hartmann solution, 214
Head, J. R.: Posture in etiology, prophylaxis and treatment of diseases of lungs, 1490
Head: See also Cranium
injury, after-effects, 741
traumatic lesions of, residual psychic phenomena following, 1458
Headaches, eyes as cause of, 201
Health Examination: See Physical examination
resorts in Harz Mountains, importance for children, 216
Hearing: See also Deafness
acuity and middle ear pressure, 455
changes in rate of human fetal heart in response to vibratory stimuli, *583
testing, methods of, 457
Heart: See also Arrhythmia; Endocarditis; Myocarditis; Pericarditis; etc.
Abnormalities: See also Heart, displacement
abnormalities; accessory ventricle, *1356
abnormalities; complete transposition of arterial trunks, *1356
abnormalities; congenital mitral malformation, 1429
abnormalities, congenital, report of 5 cases, *1356
abnormalities; double defect of ventricular septum, *1356
abnormalities; fetal extrasystoles observed in utero, 682
abnormalities; malformation with permanent slow pulse rate as result of auriculoventricular dissociation, 908
abnormalities; tetralogy of Fallot with anomalous band in right auricle, *1356
abnormalities; tetralogy of Fallot with right aortic arch and redundant left ductus arteriosus, *1356
block, congenital case of, 950
block, congenital, complete, with labile ventricular rate, case of, 937
block; double placenta praevia and prenatal diagnosis of congenital heart disease in successive labors, 681
block following lobar pneumonia, 1448
cysts; occurrence of blood-filled cysts on cardiac valves, 944
dilatation and hypertrophy, 479

Heart—Continued
disease among children of school age, 172
disease associated with congenital pulmonary cysts and hydronephrosis, 155
disease, congenital, 1428
disease; congenital lesions on pericardium, 682
disease, congenital sedimentation of blood in, 410
disease, prophylaxis of, 1192
displacement; ectopia cordis, 409
disturbances, pathology and therapy of, in dysentery, 692
Edema: See Edema, cardiac
effect of scarlet fever on, 416
electrocardiogram, influence of antirachitic therapy on, 907
fetal, changes in rate of heart beat in response to vibratory stimuli, *583
fetal, histologic investigation of muscle of, 910
gonococcic abscess of myocardium with malignant endocarditis, 1447
hypertrophy due to abnormal glycogen storage in von Gierke's disease, 704
hypertrophy, factors concerned in; study made at necropsy of 79 cases of rheumatic heart disease, 703
hypertrophy, idiopathic? 952
lymphosarcoma; specimen from girl, 955
Rate: See also Pulse
rate; cardiac malformation with permanent slow pulse rate as result of auriculoventricular dissociation, 908
rate; congenital complete heart block with labile ventricular rate; report of case, 937
rate; fatal paroxysmal ventricular tachycardia, *633
rate; paroxysmal tachycardia in child aged 7 years, 720
rate; persistent sinus tachycardia, 751
Rheumatic Disturbances: See under Rheumatic Fever
size; estimation of transverse diameter and comparison with cardiac area, 705
tuberculosis of myocardium, 437
valves; position of valves and their relation to anterior chest wall in living subjects with abnormal hearts, 703
Heat, production, predictions based on insensible loss of weight compared with direct measurements, *1300
Height; age changes in physical resemblance of siblings, 742
of oriental children in San Francisco, *1324
table of height, weight and circumference of chest, 1231
Heine-Medin's Disease: See Poliomyelitis
Helminthiasis: See Intestines, parasites
Hemangio-endothelioma of temporal bone, 438
Hemangioma, facial and intracranial, *868
results of treatment, 971
Hematocolpos: See Vagina, hematocolpos
Hemato-Encephalic Barrier: See Meninges, permeability
Hematopoietic System, Diseases: See also Anemia: Leukemia: etc.
dentistry in relation to, 707
modified technic for sternal puncture and its value in hematologic diagnosis, 942
Hemeralopia; Oguchi's disease, 1216
Hemiatrophy: See Atrophy, unilateral
Hemihypertrophy: See Hypertrophy
Hemispora stellata in ringworm of scalp, 1224
Hemochromatosis, 715
Hemoglobin: See also Anemia; Blood; Erythrocytes; etc.
formation in normal children, 483
values for average infants and children, *775
Hemography: See Leukocytes, count
Hemophilia: See also Diathesis, hemorrhagic
arrest of severe hemorrhages in all forms of hemorrhagic diathesis and in hemophilia by parenteral administration of vitamin C, 152
dental extraction in, 1443
neonatorum temporaria, fibrinogen content of blood of new-born with, 1163
Hemorrhage: See also Diathesis, hemorrhagic; Hemophilia; etc.
in New-born: See New-Born Infants, hemorrhage in
treatment with injections of artificial solutions to replace blood, 1203
Hemostasis, possibilities of snake-venom, 977
Hemotherapy in diseases of new-born with blood by mouth, 415
Hemsath, F. A.: Congenital cardiac anomalies in infants, report of 5 cases, *1356
Hepatomegaly: See under Liver
Hermaphroditism, gigantism, virilism and pseudohermaphroditism, 951
Hermetia Illucens: See Myiasis, intestinal
Hernia, diaphragmatic, 716
diaphragmatic, at esophageal hiatus, short esophagus and thoracic stomach, 680
diaphragmatic; congenital; case report, 155
diaphragmatic, congenital, rare case of, 1160
diaphragmatic, congenital, 3 cases, 409
inguinal, 1469
inguinal, in premature infants, 465
umbilical, congenital, 411
Herpes Zoster and varicella, 429
chickenpox and encephalitis, 1171
elementary bodies of zoster and their serologic relationship to those of varicella, 428
relation between virus of zoster and that of varicella, 924
Herrmannsdorfer Diet: See Tuberculosis, Gerson-Hermannsdorfer-Sauerbruch diet
Hersperger, W. G.: Mixed tumor of mediastinum of mesenchymal origin; report of case in child 3 years old, *856
Herter's Disease: See Celiac Disease
Hess, J. H.: Angiosarcoma of femur in newborn, 224
Heymann, W.: Absorption of carotene, *273
Hip Joint, congenital dislocation, diagnosis and early treatment, 1468
congenital dislocation, home of ancestors of Munich children with, 1161
congenital dislocation, roentgenologic diagnosis, 468
deformities; congenital coxa vara, 156
deformities; coxa vara at adolescence followed throughout its course, 741
roentgen study, 212
Hirschsprung's Disease: See Colon, dilatation
Histamine in relation to intradermal reaction to allergens in sensitized persons, 997
Hodgkin's Disease: See Lymphogranuloma
Hogden, C.: Metabolism of adolescent girls; excretion of creatinine and creatine, *1268
Metabolism of adolescent girls; fat and protein metabolism, *1083
Hookworm Infestation, direct development of hookworms after oral infection, 938
length of life and rate of loss of hookworms, Ancylostoma duodenale and Necator americanus, 1179
Hormones: See also Endocrine Glands; and under names of organs, as Parathyroid, hormone
glutathionemia in infancy, influence of certain hormones on, 397
in regulation of chlorides, 1156
polyhormonal syndrome in girl, 1447
Sex: See also under names of organs, as Testicles; etc.
sex: gonad-stimulating hormones from pituitary and from human urine, 439
sex: reduction of thymus by gonadotropic hormone, 146
Huff, E.: Effects on fetus of hypervitaminosis D and calcium and phosphorus deficiency during pregnancy, *302
Humidity, effect of low relative humidity at constant temperature on pollen asthma, 942
Hyaline, Degeneration: See Degeneration, hyaline
Hydatid Cyst: See Echinococcosis

Hydatid of Morgagni: See under Testicles
Hydranencephalus: See Hydrocephalus
Hydrocephalus; hydranencephaly (Cruveilhier), 227
in utero diagnosed by roentgenogram, 957
internus, disturbances of labyrinthine and optic reactions in case of, 737
otitic, 957
spontaneous perforation through large fontanel, 736
with precocious puberty following postbasic meningitis, 957
Hydrogen ion concentration, influence on activity of vaccine virus, 1168
Hydronephrosis, and cirrhosis of liver, 1453
associated with congenital pulmonary cysts and congenital heart disease, 155
Hydrophobia: See Rabies
Hydrophthalmos, congenital; rare case, 204
prognosis and treatment, 965
Hygroma: See under Bursa
Hymen, imperforate, case in which generalized peritonitis developed, 173
imperforate, hematocolpos due to, *832
imperforate, rare manifestations of, 718
Hypercholesterolemia: See Blood, cholesterol
Hyperparathyroidism: See under Parathyroid
Hyperpyrexia: See Fever
Hyperthyroidism: See under Thyroid
Hypertrophy: See also under names of organs and regions
specimens from case of right-sided hemihypertrophy associated with pubertas praecox, 1429
Hypnosis; hypnotherapy of speech disorders, 1218
in chorea minor, 195
Hypoglycemia: See Blood, sugar
Hypophysis: See Pituitary Body
Hypothyroidism: See under Thyroid
Hysterosalpingography: See under Fallopian Tubes

Ichthyosis, congenital, 160
Icterus: See Jaundice
Idiocy: See also Feeblemindedness
amaurotic, infantile, phosphatide content of brain in, 735
amaurotic, late, of Vogt-Spielmeyer type, with retinitis pigmentosa; relation to Niemann-Pick's disease, 192
amaurotic, pathogenesis, 194
mongolian, brief considerations of 4 cases, 194
mongolian, in Brazil, 1457
mongolian, in Mongolian races, 727
mongolian, results of glandular therapy in, 448
Illegitimacy, investigation of illegitimate paternity, 977
Imaginary companions, 447
Imbecility: See Idiocy
Immunity: See also Anaphylaxis and Allergy; Antigens and Antibodies; etc.
bacterial allergy and immunization process, 672
local, in subarachnoid space, 199
reactions induced in infants by intestinal absorption of incompletely digested cow's milk protein, *562
secondary cases of certain communicable diseases among nonimmune family contacts, 417
variations in communicable diseases, 694
Impetigo, bullous, followed by exfoliative dermatitis (Ritter's disease) in new-born, 1224
contagiosa, epidemiology and control, 1223
contagiosa, staphylogenous, 1224
Indians, American; inquiry on indigenous American child, 473
Infant Feeding: See also Diet; Nutrition; Vitamins; and under names of various foods, as Milk; Soy beans; etc.
acid milks in, 905
Infant Feeding—Continued
are acid whole milks to be recommended for common use in nutrition of normal infants? 152
artificial, progress in, 906
cow's milk and its derivatives, 152
feed the baby, 899
fundamentals of, 675
lactic acid milks in, 906
methods used in new-born clinic, University of Nebraska College of Medicine, for 3 years, 902
principles for, 675
prophylaxis of rickets in premature infants with vitamin D milk, *1
Infant Mortality: See also New-Born Infants; Premature Infants
native aspects of question of child care in colonies, 215
relation to normal and abnormal conditions of pregnancy and labor, 976
statistics on nursing children of society for protection of minors of Barcelona, 977
Infant Welfare: See also Child Welfare
clinic; 5 year follow-up report, 213
native aspects of question of child care in colonies, 215
station, modern concept of, 978
study of early infancy; plea for cooperation between sciences of medicine, psychology and education, 1454
Infantilism, Intestinal: See Celiac Disease
Renal: See Dwarfism, renal
Infants: See also Children; Infant Feeding; Infant Welfare; etc.
alimentary fever in infant 6 weeks old, 903
debility; relation of manganese to congenital debility, 901
Diseases: See under names of various diseases
Growth: See Growth
New-Born: See New-Born Infants
Premature: See Premature Infants
Infection: See also Bacteremia; Immunity; Septicemia; under names of bacteria; under names of organs and regions; etc.
in new-born, 682
liver extract in treatment of, 1472
resistance to, influenced by nutrition, 403
treatment with vitamin A in children with and without avitaminosis A, 1154
Infectious Diseases: See Communicable Diseases; Diphtheria; Typhoid; etc.
Influenza, antiserum; used in influenzal meningitis with recovery, *614
epidemic (1933), 685
epidemic, a virus disease, 1167
Infusions: See Injections
Injections, gangrene following subcutaneous injection of dextrose and epinephrine, 1447
intraperitoneal, accidental injury to bowel during, 707
intravenous, associated with experimental pulmonary embolism, 898
intravenous continuous, 944
intravenous; continuous method of fluid administration (venoclysis), 173
intravenous, of calcium chloride causing median nerve palsy, 191
massive infusions; experimental study, 1200
Insulin in toxic diphtheria, 920
use in nondiabetic children, 1184
Intelligence, comparative study of development in infants born in spring and of those born in autumn, 736
developmental diagnosis of infant behavior, 1233
quotient and sensitization, 994
study of 6 infant cretins; diagnostic aspects; influence of thyroid on mental growth, 1236
International Association of Preventive Pediatrics, 473
Congress for Protection of Children, next meeting, 897
Congress of Pediatrics, next session, 396, 897

Intestines: See also Colon; Duodenum; Gastro-Intestinal Tract; Intussusception; etc.
abnormalities; cysts and diverticula, *1126
absence, partial, congenital, 1430
absorption of incompletely digested cow's milk protein, as cause of immune reactions, *562
and absorption of carotene, *273
cysts, 175,
cysts and diverticula, *1126
diverticula, Meckel's, complications of, 476
diverticula, Meckel's, in infancy, 476
diverticula, Meckel's, roentgenologic diagnosis, 711
foreign bodies in, 708
Invagination: See Intussusception
motility and irritability of smooth musculature in relation to Dogiel type of intramural ganglion cell, 1151
Obstruction: See also Intussusception
obstruction, acute, 1469
obstruction due to anomalies of rotation, 464
parasites; action of copper on intestinal worms, 443
parasites; anthelmintic properties of certain alkyl phenols, 705
parasites; helminthiasis in children, 174
parasites; meningitis and meningeal reactions caused by worms, 455
parasites; pinworms and oxyuriasis, 183
parasites; worm infestation in Mecklenburg children, 1449
Stasis: See also Constipation
stasis, effect of mineral deficiencies on, 477
streptococcic, cultural and agglutinative relationships, 148
Intoxication, Intestinal: See Toxemia
Intracranial Hemorrhage in New-Born: See New-Born Infants
Intravenous Injections: See Injections, intravenous
Intubation, laryngeal, 208
Intussusception, acute, in early childhood, 1468
and intraperitoneal pressure, 974
chronic, 741
diagnosis, 1468
diagnosis and treatment by use of barium enema under fluoroscopic control, 467
early diagnosis and treatment, 1228
fluoroscopically controlled enema reduction of, 944
intestinal invagination, 976
reduction, without surgical intervention, 2 cases, 974
report of 372 cases, 463
Iodine content of potatoes, 149
review of literature on use of iodized salt in prophylaxis of goiter, 985
Ionization of nasal mucosa; relationship between reagins in blood and effect of treatment, 967
Iris, absence; inheritance and complications of congenital aniridia, 1216
Iron: calcium and phosphorus studies; effect of variation of calcium, phosphorus, and of vitamin D in diet on iron retention in rats, 401
deficiency anemia; its association with gastro-intestinal disease, achlorhydria and hemorrhage, 948
Effects: See Anemia
in Blood: See Blood, iron
influence on hemoglobin formation, 483
metabolism during pregnancy, 412
metabolism in infancy; relation to nutritional anemia, 400
quantity in placenta, 909
soluble, effect of feeding high amounts, 899
Irradiated Milk: See Milk, irradiated

Jacobsen, A. W.: Clinical results of endocrine therapy, 486
Japanese, children, growth in San Francisco, *1324
Jaundice, catarrhal, epidemic of, 1445
differential diagnosis, 1201
epidemic, in Vienna, 722
hemolytic, congenital and familial, 1194
hemolytic, congenital, mechanism of increased fragility of erythrocytes in, 703
hemolytic; mean diameter of erythrocytes in acholuric family jaundice and effects of splenectomy, 951
of new-born, *104, 684
of new-born; certain types of icterus gravis, *1059
of new-born; cholesterol of blood plasma in neonatal period, *84
of new-born, prevention by method of Keifer, 160
of new-born; van den Bergh reaction (ring test) technic and hemoglobin-bilirubin interrelation in icterus neonatorum, 910
spirochetal; meningeal form of Weil's disease without icterus and conjunctival injection, 928
spirochetal, route of infection in, 188
Jaws, osteomyelitis in new-born, 1430
Jelien, J.: Intrapericardial teratoma, *1397
Jimenez, B.: Comparative yearly surveys of specific sensitizations in students of University of Michigan from 1930 to 1935, 994
Observations on sensitization and intelligence quotient, 994
Johnson, P. A.: Immune reactions induced in infants by intestinal absorption of incompletely digested cow's milk protein, *562
Johnston, J. A.: Relationship of basal metabolism to dietary intake, *1039
Johnston, M. M.: Rôle of Lactobacillus acidophilus in production of dental caries, 478
Joints: See also under names of individual joints
ankylosis: amyoplasia congenita, 440
arthrochalasis and dermatochalasis (so-called Ehlers-Danlos syndrome), *1402
Jones, A. E.: Lesions of urinary tract, 223
Jugular Bulb: See under Veins, jugular
Jungeblut, C. W.: Blood sugar levels and dextrose tolerance in experimental poliomyelitis, *91

Kala-Azar: See Leishmaniasis
Karelitz, S.: Thrombosis of lateral sinus and jugular bulb of nonotitic origin; new diagnostic sign, *1349
Karlin, I. W.: Delay in development of speech, *1138
Kaucher, M.: Metabolism of adolescent girls; basal metabolism and energy exchange, *801
Kelly, A. D.: Cirrhosis of liver, 480
Kennedy, L.: Delay in development of speech, *1138
Keratitis, interstitial, local quinine therapy in, 1461
Keratomalacia: See Cornea, softening
Kereszturi, C.: Primary tuberculosis of skin; report of 2 cases, *847
Ketonuria: See Urine, ketone bodies
Ketosis: See Acidosis
Kidneys: See also Genito-Urinary Tract; Urinary Tract
abnormalities; congenital absence of 1 kidney with associated acute postinfectious hemorrhagic nephritis, anuria and concomitant uremia, 712
Amyloid Degeneration: See Amyloidosis
arginase with reference to age variations, 1159
carcinoma, primary, 1184
Diseases: See also Nephritis
diseases; clinical contribution to recognition and management of lipoid nephrosis, 720
diseases; lipoid nephrosis and amyloidosis in pulmonary tuberculosis, 936
diseases; nephrosis in girl of 20 months, 1446

Kidneys—Continued
diseases; observations on etiology and effect of alkalis on nephrotic syndrome, 1443
fetal, histology of, 412
function test; urea concentration range in renal inefficiency, 177
function, Volhard test in tuberculosis, 1173
Roentgenography: See Pyelography
tuberculosis, histopathology and pathogenesis of, 932
tumors, 1178, 1188
tumors, report of series of cases, 993
Kohn, J. L.: Pneumonia in children; survey of 1,000 cases with attempted follow-up, *1095
Postpneumonic residual infiltration; observations on 10 patients followed from 2 to 10 years after original admission, *1101
Koota, J.: Associated facial and intracranial hemangiomas, *868
Korsakoff syndrome in infancy, 958
Kreidel, K. V.: Blood picture after splenectomy in children with reference to platelets, *765
Kunstadter, R. H.: Medical treatment of undescended testes, 229
Kutsunai, T.: Abscess of liver of umbilical origin, report of 2 cases, *1385

Labia Minora: See Vulva
Labor, infant mortality in relation to normal and abnormal conditions of pregnancy and labor, 976
presentation; case of twins in which second fetus presented by thorax, 1430
rupture of artery of umbilical cord at beginning of, 910
Labyrinth: See Ear, internal
Lacrimal organs, structure of lower (nasal) section of nasolacrimal duct, 969
Lactation and re-onset of menstruation, 1159
consideration of milk secretion by examination of temperature of mammary gland, 906
Lactic Acid: See Acid, lactic
Lactose, effects on growth and longevity, 1419
Landry's Paralysis: See Paralysis, acute ascending
Laryngitis, obstructive panlaryngitis, 462
rare nondiphtheritic stenosis of respiratory tract, 1463
Tuberculous: See Larynx, tuberculosis
Larynx; acute infectious croup, 457
cyst, congenital, 457
cysts in new-born, 414
Inflammation: See Laryngitis
intubation, 208
papilloma, 2 case reports, 456
tuberculosis, 1440
Lateral Sinus Thrombosis: See Thrombosis
Laurence-Biedl Syndrome, case of adiposogenital syndrome with multiple malformations, 909
Laurence-Moon-Biedl Syndrome, psychologic picture of case of, 1181
Lawrence, K. B.: Mixed tumor of mediastinum of mesenchymal origin; report of case in child 3 years old, *856
Lead, classification of disease due to, 724
in peripheral blood, 30 minute spectrographic micro-method for detection of, 146
poisoning, report of case with extensive peripheral neuritis, 448
removal from drinking water, 906
Leg, fracture, intra-uterine, 1160
hypertrophy; congenital partial hemihypertrophy; report of case involving right lower extremity only, 680
Leiomyofibroma: See Fibroleiomyoma
Leishman-Donovan parasites in rhinopharynx, 1218
treatment of infantile kala-azar by intrarectal injection of tartar emetic, 441
Leishmaniasis, diagnosis by new sensitive and rapid serum reaction, 179
initial period, 178
Lens, Crystalline: See also Cataract
abnormalities; microphakia and spherophakia with glaucoma, 959
lenticonus posterior, case of, 1463
Lenticonus: See Lens, Crystalline
Lenticular Nucleus; anatomicoclinical study of pallidal syndrome, 736
Leontiasis ossium, report of case, 1203
Leslie, E. I.: Congenital thrombocytopenia, *1114
Substances involved in coagulation of blood of new-born; prothrombin; quantitative and qualitative studies of platelets in normal infant, *590
Letterer-Siwe's Disease: See Reticulo-Endothelial System, diseases
Leukemia, aleukemic, and gout in boy aged 5, 713
bone changes in, clinical and roentgenologic studies, 714
bone changes in, pathology, 714
diagnosed by supravital staining, 953
diagnostic importance of heterophile antibody test, 172
lymphatic, resembling rheumatic fever in child, 172
lymphatic; subleukemic lymphadenosis in child, 950
myelocytic, and priapism in boy aged 10 years, 719
spleen in, 1178
Leukocytes: See also Lymphocytes; etc.
Count: See also under names of various diseases, as Tuberculosis, pulmonary; etc.
count, day-to-day constancy, 176
count; hemography in diagnosis, prognosis, and treatment based on 6,000 Schilling hemograms, 708
importance of immature white blood cells in diseases of children, 947
leukopenia under physiologic and pathologic conditions, 723
leukopenic index in food allergy, 1183
monocytes in blood stream and connective tissue, 1180
monocytes in tuberculosis 1441
origin in pertussis, 1169
Leukoderma, heredity of congenital white spotting in Negroes, 970
Leukopenia: See under Leukocytes
Levine, M. I.: Tissue response of white and of Negro children to induced tuberculosis, *1052
Levine, S.: Congenital atresia of esophagus with tracheobronchial fistula, *1119
Levine, S. Z.: Creatinuria; normal variations; creatine tolerance tests and effect of aminoacetic acid in normal infants, *30
Respiratory metabolism; daily heat production of infants with predictions based on insensible loss of weight compared with direct measurements, *1300
Levinson, A.: Comparative dextrose content of lumbar and cisternal cerebrospinal fluid, *17
Encephalitis following German measles, 220
Lewis, C. A.: Relation between basal metabolism and adolescent growth, *1014
Lichen Urticatus: See Urticaria, papulosa
Life, duration, effects of lactose on, 1419
duration; foundation of longevity, 744
Light, relation between photophobia and nyctalopia, 204
report of case of meningitis due to prolonged exposure to direct rays of sun, 735
Therapy: See Ultraviolet Rays; and under names of diseases
Lilly, C. A.: Effect of generous but unfortified diet on rachitic rats, 991
Lindskog, G. E.: Changes in lung volume during treatment with artificial pneumothorax for lobar pneumonia; report of 3 cases, *523
Lipids; Lipins: See Blood, fats and lipoids

Lipodystrophy, progressive, case report, 189
Lipoma, Danlos syndrome associated with congenital lipomatosis, 211
Madelung's disease, unusual case of, 1193
Lippard, V. W.: Immune reactions induced in infants by intestinal absorption of incompletely digested cow's milk protein, *562
Lips, Harelip: See Harelip
Little, H. S.: Meckel's diverticulum in infancy, 476
Little's Disease: See Paralysis, spastic
Liver, abscess, amebic, 943
abscess, of umbilical origin, report of 2 cases, *1385
abscess, spontaneous pneumothorax and pyopericardium secondary to abscess of lung, 954
arginase with reference to age variations, 1159
cirrhosis, 480
cirrhosis, and hydronephrosis, 1453
cirrhosis, familial, 1449
cirrhosis (Laënnec) in girl of 10 years, 181
cirrhosis, portal, 1443
cirrhosis with splenomegaly, 951
extract in treatment of infection, 1472
familial hepatosplenomegaly; 2 cases in brothers, 1190
hematopoietic activity of normal and abnormal human liver, with reference to pernicious anemia, 177
hepatomegalies with disturbances of growth and metabolism of dextrose, 1194
hepatomegaly or von Gierke's disease? 952
hypertrophy; reticulo-endotheliosis, a new manifestation in hepatosplenomegalies, 186
Letterer-Siwe's disease; splenohepatomegaly associated with widespread hyperplasia of nonlipoid-storing macrophages; discussion of so-called reticulo-endothelioses, *499
vitamin A content of human liver, 1417
Logan, W. H. G.: Cleft palate and cleft lip; sequence of operations for surgical correction, 1478
Longevity: See Life, duration
Lückenschädel: See Cranium, anomaly
Lues: See Syphilis
Lumbar Puncture: See Spinal Puncture
Lungs, abnormalities; agenesis of left lung, 411
abnormalities; hypodevelopment, of lungs, case report, 442
abnormalities; vena azygos lobe in child with chronic bronchitis, 176
abscess, causation and treatment, 177
abscess, nontuberculous, treatment and management, 437
abscess, recovery, 952
abscess, roentgen studies of 2 cases, 1191
abscess with spontaneous pneumothorax, pyopericardium and liver abscess, 954
actinomycosis, 1177, 1438
collapse; atelectasis of new-born; clinical and medical legal contributions to studies of, 1163
cysts, congenital, associated with congenital heart disease and hydronephrosis, 155
cysts, congenital, diagnosis, 412
cysts, congenital, 5 cases, 174
Diseases: See also Pneumonia; Tuberculosis, pulmonary; etc.
diseases, cutaneous manifestations, relation to diagnosis and prognosis, 1448
diseases, posture in etiology, prophylaxis and treatment, 1490
diseases; survey of 1,000 cases with attempted follow-up, *1095
echinococcus cyst, 2 cases, 441
edema; acute cardiac pulmonary edema and its reflex mechanism, 185, 443
inflammation of, etiology and epidemiology, 1202
lesions associated with intra-uterine asphyxia, 157
passage of bacteria from lungs into blood stream, 149
Lungs—Continued
postpneumonic residual infiltration; observations on 10 patients followed from 2 to 10 years after original admission, *1101
stenosis, congenital, and pulmonary tuberculosis, 1440
syphilis, in child 28 days old, with observations at autopsy, 700
Tuberculosis: See Tuberculosis, pulmonary
tumor (? teratoma), 716
volume, changes during treatment with artificial pneumothorax for lobar pneumonia; report of 3 cases, *523
Lupus, disseminated, and therapeutic pneumothorax; results of heliotherapy, 933
Lymph Nodes: See also Glandular Fever; Lymphogranuloma; etc.
axillary lymphadenitis as cause of erythema nodosum, 1466
calcified abdominal glands, 980
cervical, rupture into external auditory canal, 205, 1217
diseases; clinical interpretation of lymphadenopathy, 1185
mesenteric lymphadenitis simulating acute appendicitis, 1227
submaxillary adenitis of dental origin, 182
tuberculosis; primary tuberculous cutaneous inoculation and regional adenitis treated surgically, 1439
tuberculosis; suppurative adenitis and pneumonic infiltration with favorable evolution in nursling after vaccination with B C G, 434
tuberculous, treatment of, 432
Lymphadenosis: See Leukemia, lymphatic
Lymphangioma, results of treatment, 971
Lymphatism, 1193
and rachitism, 679
clinical implications of thymus and status thymicolymphaticus, 706
Lymphocytes: See also Leukemia; Leukocytes
familial lymphocytosis, 183
Lymphogranuloma, 939, 951
diagnosis, Gordon biologic test in, 1204
malignant lymphogranulomatosis of relapsing type in girl aged 6 years, 718
malignant tumors (lymphogranulomatosis and lymphosarcoma) of mediastinum in 2 children, 1195
Lymphosarcoma: See Sarcoma, lymphosarcoma
Lysozyme, changes in lysozyme content of nasal mucus during cold, 206

McClure and Aldrich Reaction: See Edema
McCrackin, R. H.: Cystic hygroma of neck; 2 cases in Bulu children, *349
Macrostomia: See Mouth
Macula Lutea, coloboma, congenital, bilateral; familial occurrence associated with apical dystrophy of hands and feet, 961
coloboma of, so-called, 1463
familial progressive degeneration in macular region, 1215
necrosis, with perforation of retina of 1 eye and macular atrophy of other; high myopia at age of 2 years; 964
Madelung Deformity: See Hand, deformities
Malaria in infant, 687
prophylaxis, 686, 921
prophylaxis and decline in death rate in other diseases, 161
small spleen in, 912
tertian, with unusual type of skin manifestation, 421
therapeutic; dementia paralytica in girl of 13 years, treated with malaria, 1176
treatment, 690
treatment, laboratory aids in, 923
Malformation: See Abnormalities and Deformities; and under names of organs and regions
Malnutrition: See Nutrition
Malta Fever: See Undulant Fever
Mammary Gland: See Breast

Mandible: See Jaws
Manganese, relation to congenital debility, 901
relation to vitamin B deficiency, 1159
Mantoux Test: See Tuberculin Reaction
Marasmus: See under Nutrition
Marfan's Syndrome: See Fingers and Toes, abnormalities
Maroney, J. W.: Relationship of basal metabolism to dietary intake, *1039
Marples, E.: Creatinuria; normal variations; creatine tolerance tests and effect of aminoacetic acid in normal infants, *30
Mastoidectomy, diagnostic difficulties in convalescent stage, 207
temperature after, 205
Mastoiditis, complicated by hemolytic streptococcic septicemia, 461
lateral sinus thrombosis in, 458
tuberculoma of brain associated with, 453
Matheson, A.: Anthocyaninuria, 226
Maxilla: See Jaws
Maxillary Sinus: See Antrum
Measles, alveolar enanthem in, 694
classification of bronchopneumonia with reference to bronchopneumonia in measles, 689
complicated by encephalitis, 982
complicating typhoid, 688
conjunctival manifestations during incubation, 922
Dick test and measles complications, 427
early diagnosis; sign of "the eye of glass" and sign of "the semilunar fold" of E. Meyer, 688
followed by unilateral "winged scapula," 1166
German: See Rubella
immunization, 419
local skin reaction in relation to intracutaneous tuberculin reaction, 1165
or erythema infectiosum? 1171
prophylactic use of adult serum, 690, 982
prophylaxis, 690
purpuric and cerebral manifestations following; report of 2 cases, 418
rare psychic and neurologic sequels in 2 cases of encephalitis following measles, 688
serum attenuation and serum prophylaxis, 690
treatment, 918
Meckel's Diverticulum: See Intestines, diverticula
Mediastinum, cyst, 955
malignant tumors (lymphogranulomatosis and lymphosarcoma) in 2 children, 1195
tumor, mixed, of mesenchymal origin; report of case in child, *856
tumor, 2 cases, 441
Megacolon: See Colon, dilatation
Melanin, pigmentation; benign congenital melanosis of eye, 962
Melanosis: See Melanin, pigmentation
Melena vera neonatorum due to ulcus duodeni, 1165
Meninges, hemorrhage; spontaneous, case report, 452
local immunity in subarachnoid space, 199
permeability in healthy children to bromides and diphtheria antitoxin, 732
permeability; neurovegetative system; protective barriers and meningo-encephalic reaction, 731
permeability to bromide and to diphtheria antitoxin in course of diphtheria, 1434
Meningitis: See also Meningococci; Pachymeningitis
acute aseptic, 451
aseptic serous, 957
basal, followed by hydrocephalus with precocious puberty, 957
cerebrospinal, 1459
cerebrospinal, aural complications, 197
cerebrospinal; complications of, 198
cerebrospinal; ocular complications of, 197
cerebrospinal; prognosis, 198
cerebrospinal, treatment of, 958
cerebrospinal, types of meningococcus in epidemics in India, 453
Meningitis—Continued
chlorides in, 192
choriomeningitis, lymphocytic, produced in animals by newly discovered virus, 1210
dextrose content of lumbar and cisternal cerebrospinal fluid, *17
epidemic; prognosis during infancy, 196
influenzal, with recovery; use of anti-influenzal serum, *614
meningeal form of Weil's disease without icterus and conjunctival injection, 928
meningococcic and tuberculous, 193
meningococcic; Ferry's antitoxin in treatment of acute cerebrospinal fever, 1166
mumps complicated by meningitis with reference to spinal fluid, 696
pneumococcic, 1456
pneumococcic, latent symptomatologic and bacteriocytologic disassociation, 731
purulent aseptic, 730
report of case due to prolonged exposure to direct rays of sun, 735
secondary to mumps, 1169
streptococcic (viridans), with recovery, 190
tuberculous, 931, 932
tuberculous, as terminal feature in pulmonary tuberculosis, 932
tuberculous, case in infant with Nonne-Froin syndrome, 1442
tuberculous, case with no increase in cell count or globulin content of spinal fluid, 1176
tuberculous, cerebrospinal fluid in, 931
tuberculous, histopathologic observations in, 934
tuberculous, in infancy, 169
tuberculous, prevention, 165
tuberculous, primary, in parotitis in patient with tubercle bacilli in sputum, 687
tuberculous, recovery in nurslings, 167
tuberculous, seasonal, age and sex incidence, 931
tuberculous, 3 cases followed by apparent recovery, 930
tuberculous, with recovery, 433
tuberculous, with reference to racial susceptibility, 929
worms as cause of, and meningeal, 455
Meningocele, cephalic monster with unusually large meningo-encephalocele, 1209
naso-orbital, case of, 454
Meningococci carriers; high and persistent carrier rates of Neisseria meningitidis, unaccompanied by cases of meningitis, 956
types in epidemics of cerebrospinal meningitis prevalent in India, 453
Meningo-Encephalitis: See also Encephalitis; Meningitis
and acute abdominal symptoms at beginning of pneumonia, 1195
differential diagnosis of acute meningo-encephalitic processes with clear spinal fluid, 194
postpneumonic, 732
Menotoxin: See Menstruation, toxin
Menstruation, disorders; bloody tears, 201
disorders; polyhormonal syndrome, 1447
precocious, in 1 year old child with marked development of secondary sex characteristics, 1198
re-onset during lactation, 1159
toxin; allergy due to menotoxin, 221
Mental Deficiency: See Feeblemindedness
Diseases: See Nervous and Mental Disabilities
Mercury, poisoning, acute, use of sodium formaldehyde sulphoxylate in, 1471
poisoning; idiosyncrasy to ammoniated mercury ointment, 976
stomatitis due to, 942
Merritt, K. K.: Prophylaxis of rickets in premature infants with vitamin D milk, *1
Viosterol in prophylaxis of rickets in premature infants, *594

Mesentery, cyst with structure of esophagus, 1449
Lymph Nodes: See Lymph Nodes
Metabolism, 1155; See also under specific headings, as Carbohydrates, metabolism; Fat, metabolism; Mineral, metabolism; etc.
and energy exchange of adolescent girls, *801
daily heat production; predictions based on insensible loss of weight compared with direct measurements, *1300
disorders; allergy, metabolism and autonomic nervous system, 714
disturbances in relation to teeth, 674
of adolescent girls; excretion of creatinine and creatine, *1268
oxidation and radiation, 153
relation to adolescent growth, *1014
relation to dietary intake, *1039
Michigan, Children's Fund of, 396
Microcephaly; microcephalia vera, 736
Microphakia: See Lens, Crystalline, abnormalities
Microphthalmos: See Eyes, abnormalities
Milk: See also Infant Feeding
Acidification: See Infant Feeding
alimentary fever in infant due to, 903
anaphylaxis produced by, 1154
annual cycle in secretion of vitamin C by mammary gland of milch cows, 1421
antigens in, 1158
are acid whole milks to be recommended for common use in nutrition of normal infants? 152
evaporated, 1417
evaporated, in infant feeding, 1154, 1422
evils of too much milk, 902
fermentation by Mycotorulae isolated from intestines, 1155
human, as cause of malnutrition, 1159
human; calcium content after administration of calcium, viosterol and parathyroid extract, 150
human, diphtheria antitoxin in, 697
human, nonprotein nitrogen constituents, 1418
human, preservation and study of its clinical application, *249
human; relations between size and state of nutrition of mothers, properties of milk and weight of infants, 472
human; study of 45 consecutive premature infants fed on frozen mother's milk, 400
human, vitamin A in, 155
human, vitamin C content during puerperium, 1154
immune reactions induced in infants by intestinal absorption of incompletely digested cow's milk protein, *562
irradiated; influence of ultraviolet radiation on content of vitamins A, B and C of fresh cow's milk, 905
lactic acid, infant nutrition, 906
lactic acid-skimmed milk soup, 1156
pasteurization, nutritional aspects of, 899
pasteurization, time factors in, 1420
pasteurized, 1417
pasteurized, bovine tubercle bacilli in, 1177
pathologic changes in rats with anemia due to goat's milk and cow's milk, 947
pure, advantages of, in treatment of exudative diathesis, 905
vitamin D, clinical status of, 676
vitamin D, prophylaxis of rickets in premature infants with, *1
what every person should know about milk, 902
"yeast milk" and irradiated milk in infantile rickets, 1153
Mineral, deficiencies, effect on intestinal stasis, 477
metabolism, rôle in incipient tuberculosis, 1439
Mitral Valve: See under Heart
Mongolism: See Idiocy, mongolian
Monocytes: See Leukocytes
Mononucleosis, Infectious: See also Glandular Fever
heterophile antibody reaction in diagnosis of, 915
sheep and rabbit cell agglutinins in horse serum sickness and infectious mononucleosis, 674
Monsters, cephalic; unusually large meningoencephalocele, 1209
disturbances in developmental functions of germ cells; case of anencephaly, 1460
Morbidity: See Vital Statistics
Moro-Hamburger Tuberculin Reaction: See Tuberculin Reaction
Morquio's Disease: See Bones, atrophy
Mortality Statistics: See Infant Mortality; New-Born Infants, mortality; Vital Statistics
Mouth, buccopharyngeal eruption and acute pseudomembranous conjunctivitis without diphtheria, 963
histologic study of anatomic structures forming oral cavity, 672
macrostomia, unilateral, 411
rational treatment for abnormally attached fraenum labii, 1477
ulcers; case of double Riga's disease, 949
Movements, disorders; neurologic aspect of voluntary, automatic affective and spontaneous phenomena, 189
influence of disease on motor development, 453
Mucin, rôle in respiratory infections, 987
Mugrage, E. R.: Values for red blood cells of average infants and children, *775
Mumps: See Parotitis
Munson, P.: Effects on fetus of hypervitaminosis D and calcium and phosphorus deficiency during pregnancy, *302
Murphy, D. P.: Maternal age at conception of congenitally malformed child; study based on 607 cases, *1007
Muscles, abdominal, agenesia of, 155
amyoplasia congenita, 440
anatomic and pathogenic considerations of myopathies, 1455
Atrophy: See Atrophy, muscular
diseases; diagnostic and prognostic significance of creatine-creatinine metabolism in various myopathies before and after amino-acid therapy, 1178
diseases; treatment for primary myopathies, 939
Dystrophy: See Dystrophy, muscular
hypertrophy, myotonia acquisita in relation to postneuritic muscular hypertrophies, 190
inferior oblique, primary sarcoma of, 963
injuries; manipulative surgery in athletic injuries, 1228
myelomuscular dysplasia, 193
rectus abdominis, diastasis of, 1199
striated, prenatal lesions of, as cause of congenital deformity, 159
Tonus: See also Myotonia; Rigidity; and under names of muscles
Mustard packs, effect of, 474
Myatonia congenita, 955
congenita: Oppenheim's disease and early infantile progressive spinal muscular atrophy (Werdnig-Hoffmann), 1457
Mycosis, clinical case of infection with Mycotorula Pinoyi and related experimental studies, 180
fermentation of milk by Mycotorulae isolated from intestines, 1155
fungoides, ulceration of nose and face caused by, 209
Mycotorula: See Mycosis
Mydriasis: See Pupils, dilatation
Myelin, formation in prenatal infantile brain, participation of neuroglia in, 190
Myiasis, cutaneous, 1222
internal anterior ophthalmomyiasis, report of case, 1214
intestinal human, due to larvae of soldier fly, Hermetia illucens Linné (Diptera, Stratiomyidae), 938
ophthalmomyiasis subretinalis, 1460

Myocarditis, diphtheritic, 423
Myocardium: See under Heart
Myoma; adenomyoma of pylorus, 1190
Myopathy: See under Muscles
Myopia, high, at age of 2 years; macular necrosis with perforation of retina of 1 eye and macular atrophy of other, 964
muscle imbalance in, 1461
Myotonia acquisita in relation to postneuritic muscular hypertrophies, 190
congenita, 1204
congenita, family with, 1460
Myxedema: See also Thyroid, hypothyroidism
report of 2 cases to emphasize importance of bone-age studies, 941
Myxoma, mediastinal, of mesenchymal origin; report of case in child, *856

Nails, sulphur content in abnormal states, 1221
Naranjina; preparation of orange juice, 154
Nasolacrimal Canal: See Lacrimal organs
Nasopharynx, bacteriology of postnasal space, 209
National Tuberculosis Association, educational program, 666
Neck, cystic hygroma; 2 cases in Bulu children, *349
ganglioneuroma, in child, 938
Necrosis: See also Fat, necrosis; Osteochondritis; etc.
cystosteatonecrosis in new-born, 1162
Negroes, heredity of congenital white spotting in, 970
tissue response to induced tuberculosis, *1052
Neisseria: See Gonococci; Meningococci
Nemir, R. L.: Pneumonia; bacteriologic study with reference to clinical significance, *1277
Rapid typing of pneumococci by Neufeld reaction directly from laryngeal swabs, *792
Nephritis: See also under Kidneys
acute, in children, 945
Addis sediment count in juvenile nephritis with reference to effect of protein on kidneys, 989
atrophic pyelonephritis or nephritis secondary to congenital anomalies, 485
delayed rickets coexisting with chronic nephritis and glycosuria, 717
in infancy, 712
incidence and significance of minute hemolytic streptococci in, 673
pneumococcic, 1445
roentgenotherapy in, 1446
Nephrosis, Lipoid: See Kidneys, diseases
Nerves, cranial, congenital disturbances of function of motor cranial nerves, 737
facial; seventh nerve as possible pathway for transmission of virus of poliomyelitis, *58; correction, 666
optic, atrophy and bony bridges of sella turcica, 202
optic, familial hereditary atrophy, 204
optic; persistence of papilla nervi optici primitiva epithelialis, 1217
Paralysis: See under Paralysis
Nervous and Mental Disabilities: See also Chorea; Encephalitis; Idiocy; etc.
birth injuries, neurologic aspects and treatment, 414
laziness in school child, 215
mental deterioration associated with convulsions and hypoglycemia; report of 2 cases, *575
neurologic aspect of voluntary, automatic affective and spontaneous phenomena, 448
psychic "ethisms," 1457
psychopathic child in light of psychology of primitive people, 196
residual psychic phenomena following traumatic lesions of head, 1458
Nervous System: See also Brain; Cerebellum; Nerves; Reflex; Spinal Cord; etc.
autonomic, influence of diencephalon and hypophysis on general autonomic function, 448
cerebrospinal fluid in nonsyphilitic diseases of neuraxon, 958
diseases, biophysical treatment in, 453
Nervous System—Continued
heredofamilial disease of, possible explanation of dwarfism occurring in cases of, 1206
influence of disease on motor development, 453
neurologic aspect of voluntary, automatic affective and spontaneous phenomena, 189
sympathetic; allergy, metabolism and autonomic nervous system, 714
sympathetic; neurovegetative system; protective barriers and meningo-encephalic reaction, 731
Syphilis: See Syphilis
Vegetative: See Nervous System, sympathetic
Neufeld reaction, rapid typing of pneumococci by, *792
Neurinoma, origin and nature of pigmented nevi (schwannomas), 970
Neuritis, ascending, followed by encephalomyelitis disseminata, 1207
optic, acute, with sudden, transient blindness as primary symptom of acute anterior poliomyelitis; report of case, 734
peripheral, in lead poisoning, with, 448
Neuroblastoma in infant, 225
Neurofibromatosis; Recklinghausen's disease in ophthalmology, 972
Neuroglia; participation in formation of myelin in prenatal infantile brain, 190
Neuroretinitis: See Neuritis, optic; Retinitis
Neurosyphilis: See Syphilis
Neutropenia: See Angina, agranulocytic
Nevi, associated with facial and intracranial hemangiomas, *868
diagnosis and treatment, 488
naevus syringocystadenomatosus papilliferus, 971
pigmented, origin and nature of schwannomas, 970
New York State Department of Health, Streptococcus Serum: See under Streptococci, serum
New-Born Infants, 1430. See also under names of diseases, as Asphyxia neonatorum; Erysipelas; Jaundice; Melena vera neonatorum; Meningitis; Pneumonia; Syphilis, congenital
adrenal hemorrhage in normally delivered infant of diabetic mother, 683
biometric data on normal Neapolitan newborn, 1432
birth injuries, neurologic aspects and treatment, 414
birth injuries, prevention of, 1164
birth injuries; results of physical and mental training in mental deficiency caused by birth lesion, 956
birth injury; behavior aspects; neuropathology, 1234
blood calcium in, 160
blood in stools of, *255
capillaries of skin, 415
cholesterol of blood plasma in neonatal period, *84
coagulation of blood of, substances involved; prothrombin; quantitative and qualitative studies of platelets, *590
coagulation of blood, substances involved in; studies of fibrinogen, *311
coagulation of blood, substances involved in; variations in fibrinogen content in normal infant, *99
cystosteatonecrosis in, 1162
diseases, 1431
exfoliative dermatitis; epidemic pemphigus or Ritter's disease of, 684
exfoliative dermatitis (Ritter's disease) after bullous impetigo in, 1224
fibrinogen content of blood, with regard to change in hemophilia neonatorum temporaria, 1163
genitalia, physiology of, 1163
hemorrhage, intracranial, of new-born, 911
hysterosalpingography, 415
inclusion virus in eyes of new-born and in female genital tract, 966
infections in, 682
intracranial injuries, prevention of, 414
Lückenschädel of, 983

New-Born Infants—Continued
mortality, causes of, 1432
pneumothorax, spontaneous, 161
postmortem disappearance of glycogen in, 1432
precocious dentition; report of case, 682
problems of, 160
relations between size and state of nutrition of mothers, properties of their milk and weight of their infants, 472
respiration, study of rate, volume and character, *823
salaam spasms, cause of, 194
septicemia of, nasal origin in, 1162
subcutaneous adiponecrosis in, 909
treatment of diseases with human blood by mouth, 415
weight; sugar content of mother's blood after fasting; its relation to birth weight of infants, *297
Nichamin, S. J.: Hematocolpos due to imperforate hymen, *832
Niemann's Disease; late amaurotic idiocy of Vogt-Spielmeyer type, with retinitis pigmentosa; relation to Niemann-Pick's disease, 192
phosphatide content of brain in Niemann-Pick's disease and in infantile amaurotic idiocy, 735
Night-Blindness: See under Blindness
Nirvanol: See Barbital and Barbital Derivatives
Nitrobenzene poisoning, in infancy, 220
Nixon, B.: Acute appendicitis in children, *1296
Nixon, S.: Acute appendicitis in childen, *1296
Nodules, cutaneous rheumatic, 437
Noma: See Stomatitis, gangrenous
Nomland, R.: Congenital malformations of skin, including nevi; diagnosis and treatment, 488
Nonne-Froin Syndrome: See Xanthochromia
Northern Pediatric Congress, proceedings of, 1231
Nose: See also Nasopharynx
argyria nasalis, 204
clinical bacteriology of postnasal space, 209
facial and dental deformities due to perennial nasal allergy, 458
infections, concentrated streptococcus serum of New York Department of Health in, 456
ionization of nasal mucosa; relationship between reagins in blood and effect of treatment, 967
mucus, changes in lysozyme content during cold, 206
mycosis fungoides ulceration of nose, 209
nasal symptoms and reflex asthma, 175
pathogenesis of ala nasi symptom in stutterers, 968
polypi; changes in papilla of nasal origin, 202
septum; should septal resections be performed in outpatient department or in hospital? 1219
Sinuses: See Sinuses
Nucleins; nucleic acid content of thymus gland, 397
Nungester, W. J.: Rôle of mucin in respiratory infections, 987
Nutrition: See also Food; Infant Feeding; Vitamins; etc.
biochemical studies of nutritional problems, 404
disturbances; alimentary fever, 903, 1424
disturbances, ammonia and urea content and p_H of urine of healthy infants and of infants with nutritional disturbances, 151
disturbances; blood transfusion in malnutrition, 1420
disturbances, fat as genetic factor in nursling, 906
disturbances, histology of pancreas in, 1158, 1425
disturbances of nervous origin, 905
disturbances; vitamins A and D in pediatrics, 1157
Nutrition—Continued
influence on resistance to infection, 403
malnutrition and its progress in infancy, 904
work in Springfield, Illinois, schools, 402
Nyctalopia: See under Blindness
Nystagmus, monocular horizontal type, report of case, 960
nervous mechanism of, and significance in topical diagnosis of cerebral lesions, 198

Obesity, 677
and precocious general development, 716
in ossification praecox, 1452
treatment, endocrine, 486
treatment of adiposity, 1158

Obituaries:

McClure, W. B., 670
Northrup, W. P., 667

Obstetric Injury: See under New-Born Infants
Oguchi's Disease: See Hemeralopia
Oligophrenia: See Feeblemindedness
Oliver, E. A.: Dermatologic problems encountered in pediatric practice, 487
Ollier's Disease: See Achondroplasia
Ophthalmia neonatorum; pathogenicity of inclusion virus for eye of new-born and for female genital tract, 966
purulent; rôle of gonococcus in warm climates, 959
sympathetic, due to ultraviolet radiation in avitaminosis, 904
Oppenheim's Disease: See Myatonia congenita
Opsonic index in empyema, 179
Oranges, experimental study of certain preparations of orange (and of lemon) from point of view of antiscorbutic effect, 154
Orbit, osteomyelitis, fatal, 203
pseudotumor, inflammatory, 961
tumor of difficult diagnosis, 962
tumor, report of unusual case, 200
Orchitis: See Testicles, inflammation
Orthodontia: See under Dentistry
Orthopedics, progress in orthopedic surgery, 56th report, 1206
Osler's Disease: See Telangiectasis
Ossification: See also Bones, growth
praecox, 1452
Osteitis fibrosa, experimental study, 949
Osteo-arthritis, tuberculous, diagnosis of, 1441
Osteochondritis; aseptic necrosis of bones or circumscribed softening, 442
of shoulder, 1446
Osteochondroma of right femur, 719
Osteogenesis, Imperfecta: See Bones, fragility
Osteomalacia, aseptic necrosis of bones or circumscribed softening, 442
experimental, histology of cranial bones of rabbits with, 1416
Osteomyelitis, 1183
acute, 469
acute hematogenous, 1228
of maxilla in new-born, 1430
therapeutic use of concentrated antistreptococcus serum of New York State Department of Health in cryptogenic streptococcemia and osteomyelitis, 940
vertebral, 973
Osteopetrosis: See Osteosclerosis fragilis
Osteoporosis; Osteopsathyrosis: See Bones, fragility
Osteosclerosis, congenital (marble bone), 706
fragilis, 717
Otitis Media, alimentary intoxication in relation to, 739, 1190
caused by Friedländer's bacillus, symptomatology and clinical aspects, 1464
in infancy, 209, 1465
labyrinthitis and sinus thrombosis complicating suppuration of middle ear, 206
mortality in otogenic intracranial disease, 737
otitic hydrocephalus in, 957
rupture of suppurative cervical adenitis into middle ear as cause of, 1217

Otoscope, rôle of, 463
Oxygen pressure; relationship to tissue growth and glycolysis, 398
Oxyuriasis and pinworms, 183
treatment of pruritus ani in, 1449

Pachymeningitis, Hemorrhagic: See Meninges, hemorrhage
Palate, abnormalities, primary and major indications for orthodontic treatment, 1481
cleft, and cleft lip; sequence of operations for surgical correction, 1478
cleft; correcting deformities of velum and mesopharynx to insure better speech following surgery for, 466
cleft; histologic study of anatomic structures forming oral cavity, 672
cleft, treatment of, 1161
paralysis in extrafaucial diphtheria, 1166
Palsy: See Paralysis
Pancreas, cysts in region of, with notes of case, 1187
histologic investigation in nutritional disturbances, 1158, 1425
relationship between diseases of pancreas and hydrolysis of carbohydrates in feces, 153
Papilloma of larynx, 2 case reports, 456
Paralysis, acute ascending; changes in type of Landry's paralysis, *239
cerebral, 450
diphtheritic, simulated by encephalomyelitis, 1211
diphtheritic, treatment, 921
familial periodic paralysis and its transition into spinal muscular atrophy, 190
general; children of parents with dementia paralytica, 170
general; dementia paralytica in girl treated with malaria, 1176
general; hereditary syphilis, with case of juvenile paresis, 166
general; juvenile paretic neurosyphilis studies, 1436
general; juvenile paretic neurosyphilis studies; clinical course, including prodromal symptoms, nature of onset, remissions, and duration, 1436
general; juvenile paretic neurosyphilis studies; descriptive mental picture, 1435
general; juvenile paretic neurosyphilis studies; incidence; sex and age at onset, 1435
general; juvenile paretic neurosyphilis studies; treatment, 1436
Infantile: See Poliomyelitis
Landry: See Paralysis, acute ascending
median nerve palsy produced by attempted intravenous injections of calcium chloride, 191
overextension of muscles in, significance of, 735
spasmodic, late results of treatment, 1456
spastic, familial, incontinence in, 1206
spastic, treatment of, 730
Paraphrasia: See Speech, defects
Parathormone: See Parathyroid, hormone
Parathyroid, cataract in relation to, 1214
embryology and histology, 898
extract in infantile tetany; metabolic study, *816
hormone; action of parathormone (Collip) on bone tissue, 1416
hormones; distribution of calcium and phosphorus in different organs and tissues of rats in deficiency and excess of parathyroid hormone, 151
hyperparathyroidism and its relation to diseases of bone, 707, 1181
Paratyphoid and scarlet fever occurring at same time, 689
animals as carriers; significance for human beings, 162
Paresis: See Paralysis, general
Park, W. H.: Rapid typing of pneumococci by Neufeld reaction directly from laryngeal swabs, *792
Parotid Gland, Tuberculosis: See Uveoparotid fever
Parotitis and primary meningitis in patient with tubercle bacilli in sputum, 687
complicated by meningitis with reference to spinal fluid, 696
epidemic, complicated by acute appendicitis, 917
meningitis secondary to, 1169
Parrot's Disease: See Pseudoparalysis
Pasteurization: See under Milk
Patch Tests: See Anaphylaxis and Allergy
Pearlman, S. J.: Hemorrhage in course of retropharyngeal abscess, 226
Pediatric societies, directory of, 233, 493, 759, 1001, 1251, 1495
Pediatrics; Shakespeare and pediatrics, *653
surgical, 465
Pellagra and "unapparent dystrophy," 1424
etiology, 407
Pelvis, measurements of oriental children in San Francisco, *1324
Pemphigus, Epidemic: See Dermatitis exfoliativa
nonsyphilitic, congenital; review with description of new disease (epidermolysis bullosa hereditaria letalis), 1164
specificity of streptococcus isolated from patients with, 969
Penis, absence of, congenital, 680
Pentobarbital Sodium: See Barbital and Barbital Derivatives
Peppers, vitamin C in, 151
Peptic Ulcer: See also Duodenum, ulcer
in childhood, 942
in 10 year old child, 175
Percussion, new method, 217
Pericarditis, exudativa, treatment of, 1200
Pericardium, congenital lesions of heart or, 682
intrapericardial teratoma, *1397
pyopericardium, spontaneous pneumothorax, and liver abscess, secondary to abscess of lung, 954
Pericoronitis: See Gums, inflammation
Peritoneum, tuberculosis, 700, 936
Peritonitis, fetal, in premature infants, 942
generalized, in case of imperforate hymen, 173
purulent, management of, 975
Tuberculous: See Peritoneum, tuberculosis
Perlstein, M. A.: Allergy due to menotoxin, 221
Perspiration: See under Metabolism; Sweat Glands
Pertussis: See Whooping Cough
Pes Planus: See under Foot
Pharynx, buccopharyngeal eruption and acute pseudomembranous conjunctivitis without diphtheria, 963
Leishman-Donovan parasites in rhinopharynx, 12182
Phlyctenular Conjunctivitis: See Conjunctiva, tuberculosis
Phosphatase in Blood: See Blood, phosphatase
Phosphatemia: See Blood, phosphorus
Phosphorus and calcium studies; effect of prophylactic and curative doses of standardized viosterol on human tissues; necropsy report of 13 cases showing no tissue damage, 401
and calcium studies; effect of variation of calcium, phosphorus, and of vitamin D in diet on iron retention in rats, 401
distribution of calcium and phosphorus in different organs and tissues of rats in deficiency and excess of parathyroid hormone, 151
effects on fetus of hypervitaminosis D and calcium and phosphorus deficiency during pregnancy, *302
in Blood: See Blood, phosphorus
in diet of rachitic rats, 991
influence of local excess of calcium and phosphorus on healing of fractures, 464
metabolism in infantile tetany, *816
metabolism in rickets, 407
metabolism; rôle of mineral metabolism in incipent tuberculosis, 434

Photophobia: See under Light
Phrenicectomy: See under Tuberculosis, pulmonary
Physical examination; standards of physical development for reference in clinical appraisement, 471
Pick-Niemann's Disease: See Niemann's Disease
Piebald: See Leukoderma
Pincus, J. B.: Infantile tetany; metabolic study, *816
Pink Disease: See Acrodynia
Pirquet Test: See Tuberculin Reaction
Pituitary Body, adenohypophyseal dystrophies, 1189
adenomas with reference to pituitary basophilism of Cushing, 949
disorders as possible basis of dwarfism occurring in cases of heredofamilial disease of central nervous system, 1206
dyspituitarism in child, 950
gonad-stimulating hormones from pituitary and from human urine, 439
influence of diencephalon and hypophysis on general autonomic function, 448
interrelationship of adrenal cortex and anterior lobe of hypophysis, 147
physiology; gonad-stimulating hormones from pituitary and from human urine, 439
reaction of anterior pituitaries of immature female rats to injection of pregnancy urine extracts, 146
relation of experimental diabetes insipidus to anterior and posterior lobes of hypophysis, 146
rôle in thyroid syndromes, 1182
Pituitary Preparations, anterior pituitary-like principle in treatment of maldescent of testicle, 1182
posterior lobe of pituitary relationship to stomach and to blood picture, 1416
treatment of dysentery with extract of posterior hypophyseal lobe, 924
Pityriasis Lichenoides: See Erythrodermia, maculopapular
Placenta, amino-acids and iron in, quantity of, 909
and transmission of tuberculosis, 170
permeability; effects of barbiturates in embryo and on pregnancy, 742
praevia, double, and prenatal diagnosis of congenital heart block in successive labors, 681
syphilis, 933
Play, psychoanalytic theory of, 1210
Pleura, Abscess: See Empyema
Pleurisy; with serous effusion, after-history, 174
Pneumococci, history of bacteria artificially introduced into body and factors of infection; effect of cooling body on infection of Streptococcus haemolyticus-scarlatinosus and Diplococcus pneumoniae, 926
incidence of various types in infections other than pneumonia, 948
problem in children, 184
rôle of mucin in respiratory infections due to, 987
type of, and clinical significance, *1277
typing by Neufeld reaction directly from laryngeal swabs, *792
Pneumonia: See also Bronchopneumonia
bacteriologic study with reference to clinical significance, *1277
caseous, and ball-valve pneumothorax, 1172
changes in lung volume during treatment with artificial pneumothorax; report of 3 cases, *523
complicated by pneumothorax, 1180
emphysema, subcutaneous in, 954
encephalomeningitis and acute abdominal symptoms at beginning of, 1195
heart block following, 1448
in new-born, 682
in new-born; noninfectious, pneumonia of stillborn and new-born, 481
liver extract in treatment of, 1472
meningo-encephalitis following, 732
Pneumonia—Continued
postpneumonic residual infiltration; observations on 10 patients followed from 2 to 10 years after original admission, *1101
recurrence, with reference to prognosis, *284
relation to pathologic involution of thymus, 1196
serum treatment, 723
short clinical picture, 943
staphylococcic, among infants in maternity hospital, 910
survey, of 1,000 cases with attempted follow-up, *1095
tissue reactions in, 722
treatment, 1185
treatment by artificial pneumothorax, *536
typing of pneumococci by Neufeld reaction directly from laryngeal swabs, *792
Pneumothorax, artificial, changes in lung volume during treatment for lobar pneumonia; report of 3 cases, *523
artificial, in teens, 928
artificial, in treatment of lobar pneumonia, *536
Artificial, in Tuberculosis: See under Tuberculosis
ball-valve with caseous pneumonia, 1172
complicating lobar pneumonia, 1180
spontaneous, bilateral, 187
spontaneous, in new-born, 161
spontaneous, in nurslings, 1200
spontaneous, pyopericardium and liver abscess secondary to abscess of lung, 954
spontaneous tension, pneumothorax in 4 year old girl, 753
spontaneous tuberculous, 935
Poisons and Poisoning: See also under names of specific poisons, as Lead; Mercury; etc.
problem of accidents, 470
Polioencephalitis: See Encephalitis
Poliomyelitis: See also Encephalomyelitis
acute anterior, 416
acute optic neuritis with sudden, transient blindness as primary symptom; report of case, 734
autarcesiology of, 729
biophysical treatment in, 453
clinicopathologic observations; report of 125 acute cases; therapeutic use of convalescent and adult blood transfusions; possible relation of blood group to severity of disease, 725
convalescent stage, conduct of, 452
diagnosis and treatment, 728
ephedrine sulfate treatment of bulbar type, 727
experimental, alterations in mineral constituents of anterior horn cells in, 725
experimental, blood sugar levels and dextrose tolerance in, *91
immunization of experimental animals and children with formalin-inactivated virus suspension, 914
in new-born, 684
in Philadelphia, 448
mechanism of, 446
muscle compensation in, 1459
roentgen therapy in, 454
seventh nerve as possible pathway for transmission of virus, *58; correction, 666
treatment by application of short waves to spine, 454
treatment; 56th report of progress in orthopedic surgery, 1206
treatment, new, 1454
treatment with diathermy, 454
treatment with injections of antirabic virus, 689
ultrafiltration of virus, 420
use of serum and the routine and experimental laboratory findings in 1934 epidemic, 417
vaccination, successful method for, 416
virus, attempted propagation in developing egg, 397
Pollen; air studies at higher altitudes, 148
sensitiveness in children with asthma, 947
therapy, maximum dosage in, 1183

Polyarthritis: See under Arthritis
Polydactylia: See Fingers and Toes, abnormalities
Posture; education of child's body; prevention of postural deformities, 213
in etiology, prophylaxis and treatment of diseases of lung, 1490
Potassium, experimental research on relation of potassium and guanidine; pathogenesis of spasmophilia, 731
Potatoes, iodine content of, 149
Pott's Disease: See Spine, tuberculosis
Pregnancy, amino-acid in maternal blood and in that of umbilicus, 406
diet in; relation of manganese to congenital debility, 901
effect of barbiturates on embryo and on pregnancy, 742
effects on fetus of hypervitaminosis D and calcium and phosphorus deficiency, during pregnancy, *302
hemorrhage of umbilical cord, 412
hygiene; are we satisfied with results of antenatal care? 214
hygiene; effect of administration of pure vitamin A product compared with that of diet of high vitamin content, 1157
hygiene; how may health officer determine effective prenatal service? 744
hygiene; prenatal and postnatal care (2 puericultures), 978
hygiene; prenatal care, 407
hygiene; stillbirths in Meurthe-et-Moselle in past 10 years, 215
infant mortality in relation to normal and abnormal conditions of pregnancy and labor, 976
iron metabolism during, 412
maternal age at conception of congenitally malformed child; study based on 607 cases, *1007
relations between size and state of nutrition of mothers, properties of their milk and weight of their infants, 472
sugar content of mother's blood after fasting; its relation to birth weight of infants, *297
urine in; reaction of anterior pituitaries of immature female rats to injection of pregnancy urine extracts, 146
Premature Infants, calcium and phosphorus content of blood of, 1158
capillaries of, 410
congenital debility, 909
fed on frozen mother's milk, 45 cases, 400
fetal peritonitis in, 942
inguinal hernia in, 465
mortality, reduction of, 156
problem of, 158
report of 1,623 consecutive cases, 156
rickets in, viosterol in prophylaxis of, *594
rickets prophylaxis with vitamin D milk, *1
Prenatal Care: See Pregnancy, hygiene
Prepuce: See Penis
Presentation: See Labor, presentation
Preston, M. I.: Growth of oriental children in San Francisco; a contrast, *1324
Priapism associated with myelocytic leukemia in boy aged 10 years, 719
Pronation, painful, pathogenesis of: hooking of bicipital tuberosity on posterior border of lesser sigmoid cavity of ulna, 974
Protein, Addis sediment count in juvenile nephritis with reference to effect of protein on kidneys, 989
immune reactions induced in infants by intestinal absorption of incompletely digested cow's milk protein, *562
in Urine: See Albuminuria
influence of levels of protein and vitamin G, 1419
metabolism of adolescent girls, *1083
Proteose in Urine: See Urine, proteose
Prothrombin: See under Thrombin
Provitamin D: See Ergosterol, Irradiated: Vitamins, D
Prunes, vitamin B and G content of, 1419
Pruritis ani, treatment in oxyuris infection, 1449
nitrites for relief of, 1221
Pseudohermaphroditism: See Hermaphroditism
Pseudoparalysis of Parrot, 935
practical case, 935
Pseudosclerosis; sun-flower cataract in hepatolenticular degeneration; report of case, 302
Pseudotabes, postdiphtheritic, 693
Psittacosis among personnel of Hygienic Laboratory, 421
in Australian parrots, 920
Psychiatry and juvenile delinquency, 1205
is special journal for childhood psychiatry necessary? 1230
Psychology, child psychology, 726
cooperation with medicine and education in study of early infancy, 1454
primitive; psychopathic child in light of psychology of primitive people, 196
Puberty: See Adolescence
Public Health, county health work in North Carolina for 20 years, 743
district health program in rural areas, 743
Puericulture: See Child Welfare
Puerto Ricans, tissue response to induced tuberculosis, *1050
Pulse, venous, in children, 184
Pupils; anisocoria in chronic appendicitis, 1469
dilatation; mydriasis and pupillary rigidity in mother and sister with congenital syphilis, 934
pathologic retardation of absorption of pupillary membrane and its relation to other diseases, 1214
Purpura, appendicitis and acute abdominal syndrome due to, 1444
hemorrhagica; congenital thrombocytopenia, *1114
hemorrhagica, relation between purpura syndrome and tuberculosis, 700
purpuric diseases, 1450
Pyelography: intravenous urography, 719
Pyelonephritis, atrophic, or nephritis secondary to congenital anomalies, 485
Pylorus, spasms; hypertrophic pyloric stenosis, 708
stenosis, congenital, diagnosis and treatment, 1451
stenosis, congenital, hypertrophic; analysis of 145 cases treated by operation, 175
stenosis, congenital, surgical treatment, end-results, 1469
stenosis due to adenomyoma, 1190
stenosis, hypertrophic, case of 1446
stenosis; modified Rammstedt operation, 1226
Pyonephrosis, B. coli septicemia complicating pyonephrosis due to bilateral congenital ureteral strictures, 940
Pyopneumothorax and staphylococcic empyema, 1227
Pyrexia: See Fever
Pyuria: See Urinary Tract, pus in

Quadriplegia: See Extremities, paralysis
Quadruplets, monozygotic, 1162

Rabies, experimental, in white mice and attempted chemotherapy, 149
injections of antirabic virus in poliomyelitis, 689
prophylactic treatment, 1165
Race and blood groups, 1232
Rachitis: See Rickets
Radiation Therapy: See Ultraviolet Rays; and under names of various diseases
Raisins, vitamin content of sultanina (Thompson seedless) grapes and raisins, 901
Rambar, A. C.: Arachnodactylia, 225
Rammstedt Operation: See under Pylorus, stenosis
Ramon Reaction: See Diphtheria, antiserum
Rat-bite fever, 422
Ratner, B.: Pathogenesis and management of allergy in childhood, 1242
Reagins: See under Anaphylaxis and Allergy
Recklinghausen's Disease: See Neurofibromatosis

Reflex, patella, maneuver to elicit in certain cases, 958
tendon reflexes of foot, 1209
Renal Rickets: See Dwarfism
Reproduction, diet in relation to; physiologic effects of vitamin deficiencies as summarized briefly from recent experiments, 899
Resnick, R.: Blood sugar levels and dextrose tolerance in experimental poliomyelitis, *91
Respiration, breathing exercises in school, 977
rate, volume and character, study of, *823
Respiratory Tract: See also Bronchi; Nasopharynx; Nose; Pharynx, etc.
diseases; colds and allied infections, 1184
diseases; colds, changes in lysozyme content of nasal mucus during, 206
diseases, colds, new conceptions of origin and frequency of, 940
diseases; colds, vitamin "A" as prophylactic, 937
diseases; rôle of mucin in, 987
infections, acute, method of determining etiologic agent in, 1220
infections, alkali reserve and inorganic phosphorus content of blood in children with, 1424
infections, catharsis in, 706
infections; examination of children with infection of upper respiratory tract for Bacillus pertussis by cough droplet method, 421
nondiphtheritic membranous infection of lower respiratory tract, 942
roentgen study by means of injections of iodized poppy-seed oil, 1196
roentgenography (bronchography), 1219
stenosis; rare nondiphtheritic stenosis, 1463
Resuscitation: See under Asphyxia
Reticulo-Endothelial System, diseases; Letterer-Siwe's disease, 219
diseases; Letterer-Siwe's disease; splenohepatomegaly associated with widespread hyperplasia of nonlipoid-storing macrophages; discussion of so-called reticuloendothelioses, *499
reticulo-endotheliosis, a new manifestation in hepatosplenomegalies, 186
Retina: See also Macula Lutea
angiomatosis of, case report, 964
glioma, report of case with intracranial extension, 199
inflammation; late amaurotic idiocy of Vogt-Spielmeyer type, with retinitis pigmentosa; relation to Niemann-Pick's disease, 192
Retinitis, albuminuric neuroretinitis, pathologic anatomy with report of case, 203
papilloretinal exudate of dental origin, 965
Rheumatic Fever, 1186
abdominal symptoms at onset of Bouillaud's disease, 1444
cytologic studies; cells of rheumatic exudates, 673
erythema annulare rheumaticum, 228
following antidiphtheritic serotherapy, 921
gastric secretion in normal and rheumatic children, 714
heart in, 172, 946, 1182
heart in; coronary endarteritis in acute rheumatism, 713
heart in; electrocardiogram with milder degrees of chronic rheumatic heart disease, 939
heart in; factors concerned in cardiac hypertrophy; study made at necropsy of 79 cases, 703
heart in; management of children with rheumatic heart disease, report of 4 cases, 945
heart in; pericardial effusion of rheumatic origin; diagnosis and treatment, 940
heart in; sedimentation rate in rheumatic carditis, 713
heart in; social incidence of cardiac disease, statistical study in New Haven school children, 704
lymphatic leukemia resembling, 172
refractometry of blood, 724
the rheumatic child, 1189
treatment with streptococcus antitoxin, 715
tubercle bacilli as etiologic agent, 168
Rheumatic Fever—Continued
types, complications and prophylaxis in Russia, 717
Rheumatism, 948. See also Arthritis
acid metabolism in, 174
and scarlet fever, 687
cutaneous nodules, 437
etiology, 188
incidence and significance of minute hemolytic streptococci in, 673
symptoms and treatment, 438
treatment by physical methods, 1187
Rhinitis, nasal origin of septicemia in newborn, 1162
vasomotor; allergic management of, 456
Rhino-encephalocele, 725
Rickets, 903, 1422. See also Osteomalacia
avitaminosis and resistance to, 1158
craniotabes and rickets in 4 month old twins, 678
delayed, coexisting with chronic nephritis and glycosuria, 717
differentiated from juvenile osteomalacia, *618
experimental; effect of feeding high amounts of soluble iron and aluminum salts, 899
experimental; effect of generous but unfortified diet on rachitic rats, 991
experimental, histology of cranial bones of rabbits with, 1416
histophysiologic study, 679
hypervitaminosis D rickets, 677
hypervitaminosis D rickets; action of vitamin D, 404
lymphatism and, 679
neonatal, 911
phosphorus metabolism in, 407
pityriasis lichenoides et varioliformis acuta (Habermann) and, 1221
plasma phosphatase in normal and rachitic children, 1159
prophylaxis, 1157
prophylaxis, antirachitic efficacy of irradiated cholesterol, 1418
prophylaxis; antirachitic efficiency of irradiated ergosterol and cod liver oil and method for determination of rachitic weakness of legs of baby chickens, 154
prophylaxis in premature infants with vitamin D milk, *1
prophylaxis value of antirachitic agents; 5-year clinical study of, 677
prophylaxis; viosterol in rickets in premature infants, *594
Renal: See Dwarfism
spasmophilia and, 1425
spasmophilia and rickets; changes in spinal fluid in tetany, 1426
spasmophilia, in, 1427
therapy, influence shown by electrocardiogram, 907
therapy, irradiated cholesterol in, 485
therapy; parenteral administration of irradiated ergosterol, 1156
therapy, vitamin D dose required to cure and to prevent, 154
therapy; "yeast milk" and irradiated milk, 1153
types, causes of, 1422
types, clinical, 904
unusual feature of celiac disease, 954
Riga's Disease: See Mouth, ulcers
Rigidity, decerebrate, 447
Ringer's solution, modified by Hartmann in dehydration, 214
Ringworm of scalp; curability, without depilating measures, of infections caused by "animal" microsporons, 1465
of scalp, Hemispora stellata in, 1224
Ritter's Disease: See Dermatitis exfoliativa
Robertson, E. C.: Effect of mineral deficiencies on intestinal stasis, 477
Robins, L. S.: Nitrobenzene poisoning in infancy, 220
Rocky Mountain spotted fever, 698
Roehm, H. R.: Juvenile osteomalacia, *618
Roentgenotherapy: See under names of various diseases

Ronchese, F.: Dermatorrhexis with dermatochalasis and arthrochalasis (so-called Ehlers-Danlos syndrome), *1403
Rose, E.: Treatment of hyperthyroidism in children and younger adolescents, 749
Rose, E. K.: Treatment of hyperthyroidism in children and younger adolescents, 749
Rosenblum, P.: Foreign bodies in urogenital tract, 227
Roseola, exanthema subitum, 1170
exanthema subitum with encephalitic onset, 424
Ross, J.: Certain factors influencing hemoglobin formation in normal children, 483
Ross, S. G.: Certain types of icterus gravis, *1059
Rothbart, H. B.: Addis sediment count in juvenile nephritis, with reference to effect of protein on kidneys, 989
Lückenschädel of new-born, 983
Rothstein, J. L.: Calcification of choroid plexus; report of case in child aged 4 years, *123
Rubell, I.: Fatal paroxysmal ventricular tachycardia, *633
Rubella, encephalitis following, 220
Rubeola: See Measles; Rubella
Ruhräh, J., memorial to, 666

Sacks, W.: Histamine in relation to intradermal reaction to allergens in sensitized persons, 997
Interpretation of early and delayed intradermal reactions in allergic persons, 996
Sacrum, congenital absence of, 1428
partial congenital absence of sacrum and coccyx and lower sacral roots, 408
Salivary glands, pathologic changes in salivary secretion in diabetic children, 724
Sander, J. F.: Further observations on undulant fever, with report of unusual case, 979
Sanes, S.: Dilatation and hypertrophy of heart, 479
Sanford, H. N.: Congenital thrombocytopenia, *1114
Substances involved in coagulation of blood of new-born; prothrombin; quantitative and qualitative studies of platelets, *590
Substances involved in coagulation of blood of new-born; studies of fibrinogen, *311
Substances involved in coagulation of blood of new-born; variations in fibrinogen content in normal infant, *99
Sarcoma; angiosarcoma of femur in new-born, 224
lymphosarcoma involving heart; specimen from girl, 955
lymphosarcomatosis, spleen in, 1178
malignant tumors (lymphogranulomatosis and lymphosarcoma) of mediastinum in 2 children, 1195
primary, of inferior oblique muscle, 963
Sauerbruch Diet: See Tuberculosis, Gerson-Herrmanndorfer-Sauerbruch diet
Scalp, Diseases: See under names of diseases, as Ringworm; etc.
Scaphoid; congenital fusion with astragalus; bilateral; inherited, 680
Scapula, "winged," following measles, 1166
Scarlatina: See Scarlet Fever
Scarlet Fever, 418, 924. See also Streptococci, hemolytic
acetonuria in, diagnostic and prognostic significance of, 696
anatoxin, 926
antitoxin vs. no antitoxin, 419
appendicitis and acute inflammatory abdominal conditions in, 418
benzaldehyde reaction in urine in; significance in early diagnosis and differential diagnosis, 925
bronchiectasis as source of dissemination, 161
cases of both scarlet fever and disease of typhoid-paratyphoid group, 689
complicated by subacute encephalomyelitis, 922
Dick test and measles complications, 427
Scarlet Fever—Continued
emphysema, subcutaneous generalized, following bronchopneumonia and scarlet fever, 182
epidemiology, 694
erythema nodosum and, 692
heart in, 416
immunization, 431
immunization by nasal route, 422
immunization for prevention, 423
immunization in institutional epidemic, 917
immunization in prevention of specific fevers, 429
immunization, value of toxin and anatoxin of Streptococcus haemolyticus isolated from throats of patients with scarlet fever, 1168
local skin reactions in relation to intracutaneous tuberculin reaction, 1165
necrosis in throat imitating laryngeal diphtheria, 925
nucleoprotein and specific toxin derived from Streptococcus scarlatinae filtrate, 415
relapse in, 427
rheumatism and, 687
secondary case, 420
serum treatment and protective immunization, 693
suspension stability reaction (S.R.) during later course of, 1170
tongue in, 927
tonsillectomy and adenoidectomy in, 1433
toxic fractions of scarlatinal streptococci, 919
treatment, 918
vulvovaginitis due to, 1170
Scheuer, L. A.: Method of preserving breast milk; study of its clinical application, *249
Schick, B.: Essential xanthomatosis; treatment with cholesterol-free diet in 2 cases, *1372
Schick Test: See under Diphtheria
Schilling Hemogram: See Leukocytes, count
Schloss, O. M.: Immune reactions induced in infants by intestinal absorption of incompletely digested cow's milk protein, *562
Schnute, L.: Encephalitis as complication of measles, 982
Schüller-Christian Disease, case of, 186
Hand-Schüller-Christian disease and tuberculosis, 1180
ophthalmologic symptoms of, 1198
xanthomatosis and syndrome of diabetic exophthalmic dysostosis, *353
Schwannoma: See Neurinoma
Schwartz, A. B.: Rational treatment for abnormally attached fraeum labii, 1477
Sclerema: See Scleroderma
Scleroderma: lipophagic subcutaneous diffuse granulomatosis; a sclerodermic condition, 971
Sclerosis, amyotrophic lateral, mechanisms of fat metabolism in, 199
tuberous, 227
tuberous, unusual size of intraventricular spongioblastoma in case of, 1213
Scoliosis: See Spine, curvature
Scorbutus: See Scurvy
Scrofuloderm: See Skin, tuberculosis
Scurvy; ascorbic acid (vitamin C) and formation of intercellular substance in experimental scorbutus, 399
diagnosis of vitamin C subnutrition by urine analysis, 1153
effect on blood content and on urinary elimination of inorganic phosphorus in experimental scurvy, 406
experimental study of certain preparations of orange (and of lemon) from point of view of antiscorbutic effect, 154
follicular lesions in vitamin A and C deficiencies, 674
metabolism of cevitamic acid in, 1488
roentgenographic study in diagnosis of Barlow's disease, 905
Seasons; comparative study of development of intelligence of infants born in spring and of those born in autumn, 736
principal types of seasonal curve of diseases, 977

Secretions, Internal: See Endocrine Glands; Endocrine Therapy; and under names of glands
Sella turcica, bony bridges of, 202
Sensitization: See under Anaphylaxis and Allergy; Immunity; etc.
Septicemia: See also under Bacteremia; and under specific organisms and diseases
colibacillus infections, 1434
focal sepsis, 1190
following tonsillectomy, 460
hematology of infantile sepsis, 947
hemolytic streptococcic septicemia complicating mastoid disease, 461
nasal origin of, in new-born, 1162
postanginal sepsis, report of 9 cases, 913
Serotherapy: See also Hemotherapy
and hemotherapy in nursling, 1198
from hematologic point of view; study of relation between hematologic and clinical findings in diphtheria, 1434
Serum, Hanganatziu-Deicher reaction, 1199
heterospecific, electrocardiographic changes after administration of, 1199
Immune: See also Diphtheria, antiserum; Influenza, antiserum; etc.
immune, ring precipitin test for estimating concentration of antibody in small amounts of, 1151
mammalian, species nonspecific antigenic factor in, 1182
Sickness: See Anaphylaxis and Allergy
Sex, determination, reaction of Vargas for, 411
perversions; sexual psychopathologic reactions, 731
Precocious Development: See under Adolescence
Shain, J. H.: Congenital cardiac anomalies in infants, report of 5 cases, *1356
Shakespeare and pediatrics, *653
Sharp, E. A.: Status of endocrine research, 984
Shock, Anaphylactic: See Anaphylaxis and Allergy
influence of exposure to cold and of deprivation of food and water on development of, 465
Short Waves, Therapy: See Diathermy
Shoulder, osteochondritis of, 1446
Siegel, M.: Primary tuberculosis of skin; report of 2 cases, *847
Sigmoid Sinus Thrombosis: See Thrombosis
Silver; argyria nasalis, 204
Sinuses, Maxillary: See Antrum
nasal; development and lack of development of accessory nasal cavities, particularly of frontal sinus, 463
nasal sinusitis, 461
nasal; treatment of sinusitis, 455
Siwe-Letterer's Disease: See Reticulo-Endothelial System, diseases
Skin, abnormalities; Danlos syndrome associated with congenital lipomatosis, 211
abnormalities; dermatorrhexis with dermatochalasis and arthrochalasis (so-called Ehlers-Danlos syndrome), *1403
dextrose and water content of normal and of inflamed skin, 210
Diphtheria: See Diphtheria, cutaneous
Diseases: See also under names of diseases, as Eczema; Folliculitis; etc.
diseases in children, 1466
diseases, problems encountered in pediatric practice, 487
malformations, congenital, including nevi; diagnosis and treatment, 488
tuberculosis, case of scrofuloderma, 936
tuberculosis, primary, cutaneous inoculation and regional adenitis treated surgically, 1439
tuberculosis, primary, report of 2 cases, *847
tumors; dermatorrhexis with dermatochalasis and arthrochalasis (so-called Ehlers-Danlos syndrome), *1403
Skull: See Cranium
Smallpox, immunization by vaccine virus cultivated in chick embryo, 1167
inherited, 427
Vaccination: See also Encephalitis, postvaccinal
vaccination; agglutinogens of strain of vaccinia elementary bodies, 428
vaccination; flocculable substance of vaccinia, 428
vaccination; influence of *p*H on activity of vaccine virus, 1168
vaccination; maternal transmission of immunity in swine, 165
vaccination; number and length of vaccination incisions and immunity, 696
vaccination; study of vaccinal immunity by means of complement fixation, 420
Smith, C. A.: Calcified abdominal glands in children, 980
Snake-Venom: See Venom
Snelling, C. A.: Vitamin A deficiency, 484
Sobel, I. P.: Icterus gravis neonatorum, *104
Sporadic trichinosis, *367
Societies, American Orthopsychiatric Association, annual session, 145
American Public Health Association, meeting, 1150
foreign, directory of, 233, 493, 759, 1001, 1251, 1495
International Congress for Protection of Children, 897
International Congress of Pediatrics, 396, 897
international, directory of, 233, 493, 759, 1001, 1251, 1495
local, directory of, 235, 495, 761, 1004, 1254, 1498
national, directory of, 233, 493, 759, 1001, 1251, 1495
National Tuberculosis Association, educational program, 666
New England Health Education Association, meeting, 1150
Northern Pediatric Congress, sixth, proceedings of, 1231
Pediatric Association of south of Sweden, proceedings of, 1232
pediatric, directory of, 233, 493, 759, 1001, 1251, 1495
sectional, directory of, 234, 494, 760, 1002, 1252, 1496
state, directory of, 234, 494, 760, 1002, 1252, 1496

Society Transactions:

Canadian Society for Study of Diseases of Children, 475
Chicago Pediatric Society, 219, 487, 751, 1477
New York Academy of Medicine, Section of Pediatrics, 1233
Philadelphia Pediatric Society, 745, 1242, 1472
University of Michigan Pediatric and Infectious Disease Society, 979

Sodium chloride, intravenous injections in treatment of hemorrhage, 1203
thiocyanate in bacillary dysentery, 424
Solis-Cohen, L.: Congenital atresia of esophagus with tracheobronchial fistula, *1119
Sontag, L. W.: Changes in rate of human fetal heart in response to vibratory stimuli, *583
Effects on fetus of hypervitaminosis D and calcium and phosphorus deficiency during pregnancy, *302
Sound vibrations; changes in rate of human fetal heart in response to vibratory stimuli, *583
Soy beans, 150
Spasmophilia: See also Tetany
blood cholesterol in cases of epileptic convulsions and of spasmophilia, 732
pathogenesis; experimental research on relation of potassium and guanidine, 731
rickets and, 1425

Spasms, salaam spasms, cause of, 194
Speech; correcting deformities of velum and mesopharynx to insure better speech following cleft palate surgery, 466
defects; hypnotherapy of, 1218
defects; internal stuttering, 968
defects; late stuttering and late stutterers, 739
defects; nature of stuttering, 738
defects; pathogenesis of ala nasi symptom in stutterers, 968
defects; problem of stuttering, 191
defects; relation between stuttering and paraphrasia (tachylalic), 738
defects; symptomatology of stuttering, 208
delay in development of, *1138
Spekter, L.: Influenzal meningitis with recovery; use of anti-influenzal serum, *614
Spermatic cord, torsion, report of case in 10 month old infant, 709
Sperry, W. M.: Cholesterol of blood plasma in neonatal period, *84
Essential xanthomatosis; treatment with cholesterol-free diet in 2 cases, *1372
Spherophakia: See Lens, Crystalline, abnormalities
Spinal Cord: See also Meninges; Nervous System; etc.
transection from necrosis due to concussion as cause of pathothermia, 1459
Spinal Fluid: See Cerebrospinal Fluid
Spinal Puncture, accidents, acetylcholine in treatment of, 455
dextrose content of lumbar and cisternal spinal fluid, *17
safety for ambulatory patients, 447
Spine, curvature; familial scoliosis, 974
curvature; scoliosis and round shoulders, cause and treatment, 973
osteomyelitis, 973
tuberculosis, 466
Spirochaeta Gallinarum: See Spirochetosis
Spirochetosis, immunity and chemotherapy in new-born and adult animals, 1163
Spivek, M. L.: Obstructive pulmonary emphysema due to partial obstruction of bronchi by tuberculous lesions, *69
Splanchnomegaly in gigantism, 440
Spleen, cyst, 950
enlargement, and familial cirrhosis of liver, 1449
excision; blood picture after splenectomy with reference to platelets, *765
excision; results of splenectomy in Gaucher's disease, 185
Gaucher's, chemical examination of, 445
Hypertrophy: See Splenomegaly
in Hodgkin's disease, lymphosarcomatosis and leukemia, 1178
preparations; action of concentrated splenic extract in cases of certain dermatoses (eczema and urticaria) and of asthma, 970
Splenectomy: See Spleen, excision
Splenomegaly and hepatic cirrhosis, 951
differentiated from anemia, 1186
familial hepatosplenomegaly in 2 brothers, 1190
reticulo-endotheliosis, a new manifestation in hepatosplenomegalies, 186
splenohepatomegaly, associated with widespread hyperplasia of nonlipoid-storing macrophages; Letterer-Siwe's disease; discussion of so-called reticulo-endotheliosis, *499
Spohn, H.: Report of progress in cases of early celiac disease, 481
Spongioblastoma, intraventricular, unusual size of, in case of tuberous sclerosis, 1213
Sports: See Athletics
Spotted Fever: See Rocky Mountain spotted fever
Squint: See Strabismus
Stammering: See Speech, defects
Staphylococci albus; refining toxin by absorption on $Al_2(OH)_6$, 1152
aureus, pneumonia due to, among infants in maternity hospital, 910
exotoxin, ingestion by human volunteers with reference to staphylococcic food poisoning, 676
infection; infectious febrile dermatitis, 1223
is special variety of staphylococcus concerned in food poisoning? 149
Status Epilepticus: See Epilepsy
Lymphaticus: See Lymphatism
Thymicolymphaticus: See Lymphatism
Sternum, Puncture: See Bones, marrow
Still's Disease: See Arthritis deformans
Stillbirths: See also Infant Mortality
in Meurthe-et-Moselle in past 10 years, 215
noninfectious pneumonia, 481
Stokes-Adams' Disease: See Heart, block
Stomach: See also Gastro-Intestinal Tract; Pylorus; etc.
acidity; iron-deficiency anemia; its association with gastro-intestinal disease, achlorhydria and hemorrhage, 948
effect of extract of posterior lobe of pituitary gland on, 1416
emptying disturbances in, 906
hernia; thoracic, diaphragmatic hernia at esophageal hiatus and short esophagus, 680
physiology; "digestibility" of common foodstuffs as determined by radiography, 1421
roentgenography in determining "digestibility" of common foodstuffs, 1421
secretion in celiac disease, 1420
secretion in normal and rheumatic children, 714
secretion; physiology of gastric secretion; changes in relation to time factor in feeding, 153
tubercle bacilli in; examination of stomach contents for Koch bacillus; comparative value of use of stomach tube and gastric lavage, 436
Ulcer: See Peptic Ulcer
Stomatitis, aphthous, 1199
aphthous, cutaneous eruption in, 1198
Mercurial: See Mercury, poisoning
Stool: See Feces
Strabismus of unusual origin with medical cure, 964
problem of cross-eyed child, 960
treatment of, 1461
Strauss, H.: Fatal paroxysmal ventricular tachycardia, *633
Streptococci, hemolytic, and rupture of suppurative cervical adenitis into middle ear, 1217
hemolytic, effect of dissociation on specificity of, 913
hemolytic; history of bacteria artificially introduced into body and factors of infection; effect of cooling body on infection of Streptococcus haemolyticus-scarlatinosus and Diplococcus pneumoniae, 926
hemolytic, immunizing value of toxin and anatoxin isolated from throats of patients with scarlet fever, 1168
hemolytic, in throats of children in hospital wards, 456
hemolytic, minute, incidence and significance of, 673
hemolytic; observations on toxic fractions of scarlatinal streptococci, 919
hemolytic streptococcal septicemia complicating mastoid disease, 461
infections, recent wave of, 684
infections, therapeutic use of concentrated antistreptococcus serum of New York State Department of Health in cryptogenic streptococcemia and osteomyelitis, 940
intestinal, cultural and agglutinative relations of, 148

Streptococci—Continued
scarlatinae filtrate, nucleoprotein and specific toxin derived from, 415
serum; use of concentrated antistreptococcus serum of New York State Department of Health, 1180
serum; use of concentrated antistreptococcus serum of New York State Department of Health in cryptogenic streptococcemia and osteomyelitis, 940
serum; use of concentrated streptococcus serum of New York State Department of Health in infections of ear, nose and throat, 456
specificity of streptococcus isolated from patients with pemphigus, 969
viridans meningitis with recovery, 190
Stridor, congenital, due to stenosis of trachea, 1218
Strontium salts, variations in blood calcium and phosphorus after administration of, 150
Strychnine, dosage of, studies on pharmacologic and pharmacodynamic reactions, 978
ethyl alcohol and strychnine antagonism, 743
Stuttering: See Speech, defects
Subarachnoid Space: See Meninges
Subnormal Child: See Feeblemindedness
Sugar: See Carbohydrates; Dextrose; Lactose; etc.
in Blood: See Blood, sugar
Sulpharsphenamine: See also under Syphilis, therapy
death from hemorrhagic encephalitis following treatment; report of 2 cases in syphilitic infants, *609
Sulphur in hair and nails in abnormal state, 1221
metabolism in infants, 405
Summerfeldt, P.: Factors influencing hemoglobin formation, 483
Morquio's disease occurring in 2 brothers, 485
Sunlight: See Light
Suprarenal Preparations, epinephrine and dextrose, subcutaneous injection followed by gangrene, 1447
epinephrine and regulation of dextrose, 1151
epinephrine in relation to amount of water in blood, 1424
in treatment of whooping cough, 913
Suprarenals, acute insufficiency (suprarenal apoplexy, Waterhouse syndrome, Waterhouse-Friderichsen syndrome), 445
cortex, specimens from case of congenital hyperplasia, 1429
hemorrhage and subsequent calcification, 944
hemorrhage in normally delivered infant of diabetic mother, 683
interrelation of adrenal cortex and anterior lobe of hypophysis, 147
pathology; gigantism, virilism and pseudohermaphroditism, 951
sympathogonioma of, 704
Tuberculosis: See also Addison's Disease
tuberculosis in child of 12 with necropsy, *113
tumors, 723
Surgery, eosinophils after operations, 975
importance of ketosis and cyclic vomiting in, 678
in pediatrics, 465
plastic; changes in cutaneous localization in pedicle flap, 447
postoperative care of infants, 470
Sweat Glands; daily heat production; predictions based on insensible loss of weight compared with direct measurements, *1300
Sympathogonioma of adrenal gland, 704
Syndactylia: See Fingers and Toes, abnormalities
Synosteosis, Elbow: See Elbow, malformation
Syphilis: See also Paralysis, general; and under names of organs and regions, as Lungs, syphilis; Placenta, syphilis; etc.
congenital, and chronic appendicitis, 1467
congenital, and cutaneous diphtheria, report of case, 417
congenital, cerebrospinal fluid in, 1175
congenital, deformity of tibias in case of, 1439
congenital, diagnosis in neuropsychiatric disturbances, 1442
congenital, diagnosis of, 167
congenital, dystrophies and malformations of fingers in, 435
congenital, in fetus with absence of appendix, 410
congenital, late, pathologic changes in ear in, 1174
congenital, mydriasis and pupillary rigidity in mother and sister with, 934
congenital, neurosyphilis with cerebellar and pyramidal involvement, 1172
congenital, peculiar lines in hands and feet in, 1232
congenital; prematurity as etiologic factor in neonatal mortality and pathology, 164
congenital, quantitative Wassermann tests in diagnosis of, clinical importance of Fildes' law, *1257
congenital, with case of juvenile paresis, 166
Hereditary: See Syphilis, congenital
in well nourished infant, 167
juvenile paretic neurosyphilis studies, 700, 1436
juvenile paretic neurosyphilis studies; clinical course, including prodromal symptoms, nature of onset, remissions and duration, 1436
juvenile paretic neurosyphilis studies; descriptive mental picture, 1435
juvenile paretic neurosyphilis studies; incidence, sex and age at onset, 1435
juvenile paretic neurosyphilis studies; treatment, 1436
opposite behavior of monochorionic twins with reference to maternal syphilis, 436
serodiagnosis; new cataphoretic serologic reaction tested on 1,147 serums, 702
therapy: death from hemorrhagic encephalitis following treatment with sulpharsphenamine; report of 2 cases in syphilitic infants, *609
therapy, newer methods, 1172

Talipes: See under Foot
Tanaka, T.: Changes in type of Landry's paralysis, *239
Tarsorrhaphy: See Eyelids, surgery
Tears, bloody, 201
Teeth as cause of chronic submaxillary adenitis, 182
caries, among Texas school children, 214
caries, etiology of, 473
caries, rôle of Lactobacillus acidophilus in production of, 478
deformities due to perennial nasal allergy, 458
diet and nerve supply to dental tissues, 404
diseases, food as possible origin of, 977
extraction in hemophilia, 1443
formation, hereditary tendencies in, 470
hypoplasia in children of Madrid, 216
in abnormally attached fraenum labii, 1477
incipient orthodontic needs, 1470
infected, as cause of papilloretinal exudate, 965
metabolic disturbances in relation to, 674
mottled, experimental and histologic analysis of, 1229
movement of tooth germ through jaw, 471
of cement workers, 474
precocious dentition in new-born; report of case, 682
primary and major indications for orthodontic treatment, 1481

Teeth—Continued
public school dental clinic, 11 years' work in, 214
tooth in lid and palpebral coloboma, 201
vitamins in relation to normal growth and dentition, 901
Telangiectasis, familial hemorrhagic angiomatosis (Osler's disease), 1223
Temperature, Body: See also Fever
disturbances of, 1188
neurogenic and unconstitutional components in origin of protracted subfebrile temperature, 186
variations, 183
Temporal bone, hemangio-endothelioma of, 438
Teratoma, abdominal, 1447
abdominal; report of series of cases, 993
mediastinal, of mesenchymal origin in child, *856
neoplasm of lung (? teratoma), 716
Terplan, K.: Incidence and types of tuberculosis in children, 475
Testicles, inflammation; acute orchitis in infancy; torsion of hydatid of Morgagni, 720
maldescent of, anterior pituitary-like principle in treatment of, 1182
undescended, hormone and operative treatment of, 1162
undescended, in man and monkeys, 171
undescended, medical treatment of, 229
Tetanus, combined active and passive immunization against diphtheria and tetanus, 688
researches; toxin of Bacillus tetani is not transported to central nervous system by any component of peripheral nerve trunks, 146
toxoid immunization, 420
treatment, 426, 430, 1170
without discernible wound; intensive serum therapy with cure, 1167
Tetany, cerebrospinal fluid changes in, 1426
epilepsy and cataract, 193
in infants aged less than 3 months, 199, 730
infantile, metabolic study, *816
low calcium, in new-born, 727
of new-born, 912
of new-born with deposit of calcium following parenteral administration, 219
physicochemical study of blood of 10 adults presenting Chvostek sign, 1455
prophylaxis, 1157
rickets with and without symptoms of spasmophilia, 1427
Therapeutics, use and abuse of drugs, 1471
Thiobismol, fatility from exacerbation of latent tuberculosis due to, in case of yaws, 1437
Thomsen's Disease: See Myotonia congenita
Thorax; nonrachitic soft chest, 187
table of height, weight and circumference of chest, 1231
Throat, infections, concentrated streptococcus serum of New York State Department of Health in, 456
Streptococcus hemolyticus in throats of children in hospital wards, 456
Thrombin, substance involved in coagulation of blood of new-born; prothrombin; quantitative and qualitative studies of platelets, *590
Thrombocytopenia: See Purpura hemorrhagica
Thrombopenia: See under Diathesis, hemorrhagic
Thrombophlebitis of lateral sinus and jugular bulb of nonotitic origin, new diagnostic sign, *1349
Thrombosis, lateral sinus and jugular bulb, of nonotitic origin; new diagnostic sign, *1349
lateral sinus, complicating mastoiditis, 458
lateral sinus, report of 30 cases, 455
sinus, and labyrinthitis complicating suppuration of middle ear, 206
Thumb: See Fingers
Thumbsucking: See Fingersucking
Thymoma, malignant, 222
Thymus, atrophy in renal rickets, 1203
autoplastic transplantation in fowl, 397
cyst in new-born, 715
embryology and histology, 898
hypertrophy differentiated from congenital atresia of esophagus with tracheobronchial fistula, *1119
influence on behavior of phosphatemia and glycemia, 406
involution of, and its relation to pneumonia, 1196
malignant thymoma, 222
nucleic acid content, 397
reduction by gonadotropic hormone, 146
Status Lymphaticus: See Lymphatism
weight; component parts and number of Hassall corpuscles, *313
Thyroid, Diseases: See also Goiter
diseases, treatment of, 1450
disturbances, rôle of hypophysis in, 1182
hyperthyreosis, 1453
Hyperthyroidism: See also Goiter
hyperthyroidism, treatment of, 749
Hypothyroidism: See also Cretinism; Myxedema
hypothyroidism; blood cholesterol and creatine excretion in urine as aids to diagnosis and treatment, 1179
hypothyroidism; clinical results of endocrine therapy, 486
hypothyroidism, slight signs of, 1445
influence on behavior of phosphatemia and glycemia, 406
influence on mental growth, 1236
surgery, indictions for, 466
Tibia, deformity in case of hereditary syphilis, 1439
diseases; achondroplasia confined to half the upper end of tibia; rapid evolution in 12 year old girl, 409
Tisdall, F. F.: Irradiated cholesterol in rickets, 485
Rôle of Lactobacillus acidophilus in production of dental caries, 478
Tissue, chemistry; new method for obtaining tissue fluid, 186
connective, behavior of monocytes in, 1180
growth, relation to glycolysis and oxygen pressure, 398
reactions in pneumonia, 722
Toes: See Fingers and Toes
Tomato Juice, canned, vitamin C content, 402
vitamin G content of home-canned juice, 901
Tongue, cyst, congenital glosso-epiglottic, 967
Tonsillectomy, anesthesia, local, review based on 500 cases, 460
complications and results, 460
followed by septicemia, 460
for peritonsillar abscesses, 1220
in scarlet fever; survey, 1433
should tonsillectomies and septal resections be performed in outpatient department or in hospital? 1219
ulcus vulvae in child following, 208
Tonsillitis, clinical and pathologic aspects of chronic tonsillitis and of chronic irritation of tonsillar crypts, 738
Tonsils, Leishman-Donovan parasites in rhinopharynx, 1218
Toomey, J. A.: Seventh nerve as possible pathway for transmission of virus of poliomyelitis, *58; correction, 666
Torula, cutaneous torulosis, 1221
Towsley, H.: Review of literature on use of iodized salt in prophylaxis of goiter, 985
Toxemia, alimentary, and otitis media in nurslings, 1465
alimentary, case of, 407
alimentary, chronic, 903
alimentary; diminution of blood volume in infantile intoxication, 907
alimentary, in relation to otitis media, 1190
alimentary; old and new ways in management of toxicosis, 1156

Toxemia—Continued
alimentary, otitides of infants and their relation to, 739
alimentary; therapy of intoxication and diarrhea, some new points of view, 1152
Trachea, Fistula: See Fistula
Foreign Bodies: See Foreign Bodies
stenosis, congenital stridor due to, 1218
stenosis, rare cases of, 462
story of tracheotomy, 207, 458
Tracheotomy: See under Trachea
Trachoma, prophylaxis of, 965
Transfusion: See Blood, transfusion
Trichinosis, sporadic, *367
Trichotillomania: See Hair, plucking
Trypanosomiasis, first fatal case of Chagas' disease observed on Isthmus of Panama, 938
Tubercle Bacilli; bactericidal power of blood serum, 435
bovine, in pasteurized milk and butter, 1177
cultivation, difficulties with acid-fast saprophyte, 1178
examination of stomach contents for Koch bacillus; comparative value of use of stomach tube and gastric lavage, 436
free growth period in guinea-pig omentum as related to hypersensitive state, 431
in blood and spinal fluid in chorea, 183
in blood; cultured by method of Löwenstein, 168
in blood; tubercle bacillemia in course of acute polyarthritis, 1174
Tuberculin Reaction; allergy and ocular phlyctenulosis, 1215
comparative study of tuberculin tests, 166
cutaneous reactions with avirulent tuberculin, 1173
difficulties in diagnosis of, 1173
effect of vaccination with B C G in tuberculosis, with correlation of tuberculin reactions, roentgen diagnosis and mortality, 748
evaluation of skin tests in school health survey, 432
in infants and small children, 434
intracutaneous, in relation to local skin reactions in measles and scarlet fever, 1165
intracutaneous, relation to physical constitution of Korean school children, 702
mechanism of tuberculin hypersensitiveness, 930
Loewenstein, 170
Mantoux intracutaneous reaction, comparative study of positive and negative reactors, 1436
Mantoux tests in general hospital population, 933
Moro-Hamburger reaction, value of, 168
Pirquet reaction, difference, on trunk and limbs, 701
sensitivity of children having slowly ingested BCG vaccine, 1172
sensitivity of skin in primary tuberculosis, 164
studies based on clinical material of hospital; practical value of various tuberculin tests, 1175
Tuberculoma of brain associated with ear disease, 453
Tuberculosis, 930, 1177, 1437: See also under names of organs and regions, as Bronchi, tuberculosis; Lymph Nodes, tuberculosis; Skin, tuberculosis; Suprarenals, tuberculosis; etc.
annual review, 433
as factor in calcified abdominal glands, 980
BCG; afebrile pulmonary infiltrations as symptoms of bécégite in infants at birth, 433
BCG, effect of vaccination, with correlation of tuberculin reactions, roentgen diagnosis and mortality, 748
BCG, effect on infant mortality, 1173
BCG, immunization by vaccination with Calmette-Guérin bacillus, 702
Tuberculosis—Continued
BCG, lesions in rabbits following inoculation with, 929
BCG, peroral vaccination with BCG in Norbotten, 1177
BCG, stability of colony morphology and pathogenicity of BCG, 929
BCG; suppurative adenitis and pneumonic infiltration with favorable evolution in nursling after vaccination with BCG, 434
BCG; tuberculosis in young children receiving BCG, 1177
BCG vaccine, tuberculin sensitivity of children having slowly ingested BCG vaccine, 1172
bacteremia; tubercle bacilli in blood and spinal fluid in chorea, 183
blood in; monocytes, 1441
blood pressure of normal and tuberculous children, 435
bovine, in relation to man, 433
climatic therapy in asthenic conditions in infancy, 435
congenital, case of, 1439
control, 1437
diagnosis in infants, 169
erythema nodosum and, 1176, 1442
Gerson-Herrmannsdorfer-Sauerbruch diet, 1440
in twins, differently nurtured, 1442
incidence and types, 475
influence of varicella on, 935
inoculation with heat-killed human tubercle bacilli, 165
kidney function test in, 1173
latent, fatality from exacerbation due to thiobismol in case of yaws, 1437
marriage in, 934
Meningitis Due to: See Meningitis, tuberculous
miliary; healed (calcified) miliary tuberculosis of lungs, 166
mineral metabolism in incipient cases, 434, 1439
osteo-articular, diagnosis of, 1441
placental transmission of, 170
primary, cutaneous inoculation and regional adenitis treated surgically, 1439
prophylaxis, 1441
pulmonary; afebrile pulmonary infiltrations as symptoms of bécégite in 2 infants vaccinated with BCG at birth, 433
pulmonary and congenital pulmonary stenosis, 1440
pulmonary, and Hand-Schüller-Christian disease, 1180
pulmonary, cavities in, 701
pulmonary; cholesterol content of blood of tuberculous children after sojourn at high altitude, 168
pulmonary; disseminated lupus and therapeutic pneumothorax; results of heliotherapy, 933
pulmonary, early manifestations in Texas children, 432
pulmonary; healed (calcified) miliary tuberculosis, 166
pulmonary, huge tension cavity in infant, 165
pulmonary, in infants, 431
pulmonary, leukocyte picture in, with study of filament-nonfilament count, 164
pulmonary, lipoid nephrosis and amyloidosis in 936
pulmonary, meningeal tuberculosis as terminal feature in, 932
pulmonary, new orientations in study of, 168
pulmonary, phrenicectomy in, 934
pulmonary; primary tuberculosis, obstructive pulmonary emphysema and asthma with recovery, *118
pulmonary, prognosis, 434
pulmonary; results of bilateral therapeutic pneumothorax for ulcerative tuberculosis of left lung and infiltration of right; cure after 5 years of treatment, 933
pulmonary spontaneous pneumothorax, 935

Tuberculosis—Continued
pulmonary; what does primary tuberculous infection prevent? 165
pulmonary; why pulmonary form of tuberculosis is common in adults and nonpulmonary form in children, 1438
purpura syndrome in relation to tuberculosis, 700
Skin: See Skin
tissue response of white and of Negro children to induced tuberculosis, *1052
Tuberculin Reaction: See Tuberculin Reaction
urine in, studies in vitro of specific antigen, 935
vaccine therapy, 166
vaccine therapy; interesting clinical case of alphatherapy, 701
viosterol in, effect of moderately large doses, 163
vitamin deficiency and, 701
whooping cough and, 1171
why pulmonary form is common in adults and nonpulmonary form in children, 1438
Tumors: See also Adenoma; Fibroleiomyoma; Sarcoma; etc.; and under names of organs and regions, as Femur, angiosarcoma
solid, puncture biopsy as diagnostic measure in differentiation of, 986
Tumpeer, I. H.: Tetany of new-born with deposit of calcium following parenteral administration, 219
Twins, case in which second fetus presented by thorax, 1430
craniotabes in 4 month old twins, 678
Huntington's chorea in, 1206
identical, epilepsy in 3 pairs of, 1208
inheritance of diabetes in, 1182
monochorionic; opposite behavior with reference to maternal syphilis, 436
psychologic picture of case of Laurence-Moon-Biedl syndrome in a twin, 1181
refractive error of, 962
studies in infancy and preschool age periods, 218
tuberculous infection in pair of differently nurtured twins, 1442
Typhoid, alkaline treatment, 424
Bretonneau and first French literature on dothienenteritis, 928
complicated by measles, 688
complicated by scarlet fever, 689
in vaccinated men, 425
Typhus, control in public schools in city of Lodz, 926

Ulcers: See under names of organs and regions
Peptic: See Peptic Ulcer
Ulna, pathogenesis of painful pronation hooking of bicipital tuberosity on posterior border of lesser sigmoid cavity of ulna, 974
Ultraviolet Rays and metabolic oxidation, 153
influence on time of absorption of provoked edema and on activity of edema-producing substances, 398
sympathetic ophthalmia due to, in avitaminosis, 904
Therapy: See under names of various diseases, as Erysipelas; etc.
Umbilical Cord; amino-acid in maternal blood and in that of umbilicus, 406
circulation, exchange of lipids in, at birth, 900
hematoma, case report, 161
intra-uterine hemorrhage, 412
rupture of artery of, at beginning of labor, 910
Umbilicus, abscess of liver of umbilical origin, report of 2 cases, *1385
Uncinariasis: See Hookworm Infestation
Undulant Fever, intradermal test, 421
observations on, with report of unusual case, 979
report of cases, 418
Urea, concentration range in diagnosis and prognosis of kidney inefficiency, 177
Ureters, ectopia, incontinence of urine due to, 157
strictures; B. coli septicemia complicating pyonephrosis due to bilateral congenital ureteral strictures, 940
Urethra, valvular obstruction associated with renal dwarfism, 951
valvular obstruction in prostatic urethra, congenital, 158
Urinary Tract: See also Genitals; Genito-Urinary Tract; Kidneys; Ureters; etc.
colon bacillus infection, 1169
infection, ketosis and ketogenic diet in, treatment of, 1207
lesions, 223
pus in; chronic pyuria (congenital defect); presacral sympathectomy, 466
pus in; study of 40 cases of persistent pyuria, 943
pus in, treatment, 1447
Urination, incontinence due to ureteral ectopia, 157
incontinence; enuresis, statistical and analytic study, 1476
incontinence in familial spastic paralysis, 1206
incontinence, nocturnal, 175, 439
Urine: See also Pregnancy, urine in; Urination
Acetone in: See Urine, ketone bodies
Albumin in: See Albuminuria
ammonia and urea content and *p*H of urine of healthy infants and of infants with nutritional disturbances, 151
ammonia coefficient of, 406
analysis in diagnosis of vitamin C subnutrition, 1153
anthocyaninuria, 226
auto-injection in allergic conditions, 184
bilirubin in, simple and exact test for, 1417
colibacillus in, 1434
creatine and creatinine excretion of adolescent girls, *1268
creatine in, normal variations; creatine tolerance tests and effect of amino-acetic acid in normal infants, *30
creatine excretion and blood cholesterol in urine as aids to diagnosis and treatment of hypothyroidism, 1179
creatine-creatinine metabolism in various myopathies before and after amino-acid therapy, diagnostic and prognostic significance, 1178
examination; Addis sediment count in juvenile nephritis, with reference to effect of protein on kidneys, 989
ketone bodies; diagnostic and prognostic significance of acetonuria in scarlet fever, 696
phosphorus; effect on blood content and on urinary elimination in experimental scurvy, 406
proteose in eczema, 210
sugar; delayed rickets coexisting with chronic nephritis and glycosuria, 717
sugar; renal dwarfism and glycosuria, 955
Urogenital Tract: See Genito-Urinary Tract
Urography: See Pyelography
Urticaria, concentrated splenic extract in, 970
papulosa; lichen urticatus, 1222
review of 170 cases of urticaria and angioneurotic edema followed for from 2 to 10 years, 211
Uveoparotid fever; tuberculosis of parotid gland, 936

Vaccination, Encephalitis Following: See under Encephalitis
Vaccine, Ferrán's: See Tuberculosis, vaccine therapy; Whooping Cough
Therapy: See under specific headings, as Diphtheria; Tuberculosis; Whooping Cough; etc.
Vaccinia: See Smallpox, vaccination
Vagina, foreign bodies in, simplified method of removal with report of 3 cases, 940
hematocolpos due to imperforate hymen, *832

Vaginitis, etiology of vulvovaginitis, 1170
gonorrheal, amniotin in, 171
gonorrheal, diathermy in, 944
gonorrheal, etiology of, 1170
gonorrheal; lyzed gonococci in treatment of cervicovaginitis, 440
gonorrheal, new treatment of, 1166
gonorrheal, pyridium in treatment of, 439
Vargas reaction for determination of sex, 411
Varicella: See Chickenpox
Variola: See Smallpox
Veeder, B. S.: Special report of the American Board of Pediatrics, Inc., 389
Veins, jugular, thrombosis of nonotitic origin; new diagnostic sign, *1349
pulmonary, left, leiomyofibroma of, 1196
Venereal Diseases: See Gonorrhea; Syphilis
Venoclysis: See Injections, intravenous
Venom, snake, hemostatic possibilities of, 977
Ventriculography: See Brain, roentgenography
Verruca, review of, 970
Vertebrae: See Spine
Vincent's Angina: See Angina
Vinograd, J.: Pneumonia; bacteriologic study with reference to clinical significance, *1277
Rapid typing of pneumococci by Neufeld reaction directly from laryngeal swabs, *792
Viosterol: See Ergosterol, Irradiated
Viruses, lymphocytic choriomeningitis produced in experimental animals by newly discovered virus; studies of origin of virus, 1210
Vision, defects, prevalence in children; eye survey at St. Christopher's Hospital, 200
refractive error of twins, 962
relation between photophobia and nyctalopia, 204
testing eyes of school children; new test cabinet, 959
Vital Statistics, development of pathologic differences in relation to age, 474
morbidity in family tree, 472
Vitamins: See also Cod Liver Oil; Deficiency Diseases; Milk; Rickets; etc.
A and D in pediatrics, 1157
A as prophylactic against common "cold" in school children, 937
A, content of human liver, 1417
A deficiency, 484
A deficiency, follicular lesions in, 674
A deficiency, parenteral use of carotene in treatment of, in albino rats, 676
A deficiency; xerosis in eye clinic, 1462
A; diet and nerve supply to dental tissues, 404
A; effect during pregnancy of administration of pure vitamin A product compared with that of diet of high vitamin content, 1157
A, effect of carotene on, 150
A in blood serum and in mother's milk, 155
A in cure of various infections with and without avitaminosis A, 1154
A, influence of massive doses on animals deprived of same in diet, 1422
A, rôle in nutrition, 902
A, specific effect on growth, 403
B and G content of prunes, 1419
B deficiency, relation of manganese to, 1159
B, sparing action of fat on vitamin B: influence of levels of protein and vitamin G, 1419
B; sparing action of fat on vitamin G, 1419
B; vitamin G content of home-canned tomato juice, 901
C, 1160
C, arrest of severe hemorrhages in all forms of hemorrhagic diathesis and in hemophilia by parenteral administration of vitamin C, 152
C (ascorbic acid) isolated from Capsicum annuum, 151
C; cevitamic acid, 405
C, cevitamic acid; color reaction for ascorbic acid, 398
C content of canned tomato juice, 402
Vitamins—Continued
C content of human mammary secretion during puerperium, 1154
C, deficiency, capillary resistance test as measurement of, 478
C, deficiency, follicular lesions in, 674
C subnutrition, diagnosis by urine analysis, 1153
C, treatment of hemorrhagic diathesis, more especially essential thrombopenia with, 1460
content of figs, 901
content of sultanina (Thompson seedless) grapes and raisins, 901
D and A in pediatrics, 1157
D content of egg yolk, 402
D, dose required to cure and to prevent rickets, 154
D; effect of variation of calcium, phosphorus and vitamin D in diet on iron retention in rats, 401
D; effect on fetus of hypervitaminosis D and calcium and phosphorus deficiency during pregnancy, *302
D; hypervitaminosis D rickets, 677
D in treatment of eczema, 1217, 1463
D milks, clinical status of, 676
D; provitamin D of cholesterol; antirachitic efficacy of irradiated cholesterol, 1418
D, studies 1933-1934, 400
Deficiency: See also Beriberi; Deficiency Diseases; Rickets; Scurvy; etc.; and under specific vitamins
deficiency and tuberculosis, 701
experimental study of certain preparations of orange (and of lemon) from point of view of antiscorbutic effect, 154
F: See Acid, fatty
G: See Vitamins, B
in relation to normal growth and dentition, 901
Reproduction and Vitamins: See under Reproduction
Volhard Test: See Kidneys, function
Vomiting and loss of appetite of child, 407
cyclic, surgical importance of, 678
in early infancy, 679
recurrent attacks, 677
Vulva, adhesions of labia minora, 1200
Diphtheria: See Diphtheria, vulvovaginal
ulcus vulvae following tonsillectomy, 208
Vulvovaginitis, Diphtheritic: See Diphtheria, vulvovaginal
dysenterica, 693
etiology of, 1170

Wallace, R. F.: Changes in rate of human fetal heart in response to vibratory stimuli, *583
Wang, C. C.: Metabolism of adolescent girls; basal metabolism and energy exchange, *801
Metabolism of adolescent girls; excretion of creatinine and creatine, *1268
Metabolism of adolescent girls; fat and protein metabolism, *1083
Warts: See Verruca
Warwick, M.: Noninfectious pneumonia of stillborn and new-born infants, 481
Wassermann Reaction: See under Syphilis
Water, drinking, removal of lead and copper from, 906
metabolism in diphtheria (reaction of McClure and Aldrich), 162
Waugh, T. R.: Certain types of icterus gravis, *1059
Weight, age changes in physical resemblance of siblings, 742
of New-Born: See under New-Born Infants
of nondiabetic children treated with insulin, 1184
of oriental children in San Francisco, *1324
studies on growth and economic depression, 677
table of height, weight and circumference of chest, 1231
Weil's Disease: See Jaundice, spirochetal

Weiner, S. B.: Pneumonia in children; survey of 1,000 cases with attempted follow-up, *1095
Weinfeld, G. F.: Persistent sinus tachycardia, 751
Weiss, S.: Enuresis; statistical and analytic study, 1476
Welander's ulcer, 1452
Weld, C. B.: Capillary resistance test as measurement of vitamin C deficiency, 478
Werdnig-Hoffmann's Disease: See Atrophy, muscular
Wheatley, M. A.: Respiratory metabolism; daily heat production of infants with predictions based on insensible loss of weight compared with direct measurements, *1300
Whooping Cough, bacilli; examination of children with infection of upper respiratory tract for Bacillus pertussis by cough droplet method, 421
bacteriologic diagnosis, 1432
diagnosis and therapy, 924
diagnosis without whoop, 429
early diagnosis by simplified cough-plate method, 426
early diagnosis, prevention and treatment, 426
Haemophilus pertussis endoantigen (Krueger) in prevention and treatment, 423
in adults, 423
leukocytosis in, origin of, 1169
problem, 698
statistical analysis of cases, 422
toxin of Bacillus pertussis, 697
treatment, 918, 923
treatment with bromides, 925
treatment with suprarenal preparation and serum, 913
tuberculosis and, 1171
vaccination, prophylactic, 162
vaccination, statistics on, 691
vaccine, 685
vaccine; clinical notes on use of anti-alpha vaccine, 691
vaccine, prophylactic, 419, 1473
vaccine; prophylactic use of pertussis vaccine (Sauer), 418
vaccine, results of use of, 698
vaccine treatment, 694
Wile, S. A.: Coarctation of aorta, 224
Wilm's Tumor: See Embryoma
Winckel's disease, severe case, 911
Wing, M.: Metabolism of adolescent girls; basal metabolism and energy exchange, *801
Metabolism of adolescent girls; fat and protein metabolism, *1083
Wolf, A.: Xanthomatosis and syndrome of diabetic exophthalmic dysostosis, *353
Wollstein, M.: Blood picture after splenectomy in children with reference to platelets, *765
Woods, E. B.: Sugar content of mother's blood after fasting; its relation to birth weight of infants, *297
Worms: See Intestines, parasites
Wortis, S. B.: Xanthomatosis and syndrome of diabetic exophthalmic dysostosis, *353
Wrist, circumference, as indication of bone growth, 1231

Xanthochromia, case of tuberculous meningitis with Nonne-Froin syndrome, 1442
Xanthoma; essential xanthomatosis, treatment with cholesterol-free diet in 2 cases, *1372
generalized xanthomatosis, 184
Xanthomatosis: See Anemia, splenic; Niemann's Disease; Schüller-Christian Disease; Xanthoma
Xerophthalmia, experimental, favored by carbohydrates? 405
influence on rate of blindness, 1216
treatment; new indication for central tarsorrhaphy, 962
xerosis in eye clinic, 1462
Xerosis: See Xerophthalmia

Yaws: See Frambesia
Yeast, irradiated milk and yeast milk in infantile rickets, 1153

Zeitschrift für Kinderpsychiatrie; Is special journal for childhood psychiatry necessary? 1230
Zimmerman, H.: Case of injury at birth; neuropathology, 1234
Zona; Zoster: See Herpes Zoster

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CONTENTS

PAGE

Quantitative Wassermann Tests in Diagnosis of Congenital Syphilis: Clinical Importance of Fildes' Law. Harold K. Faber, M.D., and William C. Black, M.D., San Francisco....1257

Metabolism of Adolescent Girls: III. The Excretion of Creatinine and Creatine. Chi Che Wang, Ph.D.; Ida Genther, Ph.D., and Corinne Hogden, M.S., Cincinnati....1268

Pneumonia in Infants and in Children: A Bacteriologic Study with Special Reference to Clinical Significance. Rosa Lee Nemir, M.D.; Elizabeth Torrey Andrews, M.D., and Julia Vinograd, M.S., New York....1277

Acute Appendicitis in Children. Sater Nixon, M.D., and Byron Nixon, M.D., Indianapolis....1296

Respiratory Metabolism in Infancy and in Childhood: XVII. The Daily Heat Production of Infants—Predictions Based on the Insensible Loss of Weight Compared with Direct Measurements. S. Z. Levine, M.D., and M. A. Wheatley, M.D., New York....1300

Growth of Oriental Children in San Francisco: A Contrast. Mary I. Preston, M.D., San Francisco....1324

Thrombosis of the Lateral Sinus and Jugular Bulb of Nonotitic Origin: A New Diagnostic Sign. Samuel Karelitz, M.D., New York....1349

Congenital Cardiac Anomalies in Infants. Frederick A. Hemsath, M.D., Wilmington, Del.; Morris Greenberg, M.D., New York, and Joseph H. Shain, M.D., Boston....1356

Case Reports:

Essential Xanthomatosis: Treatment with Cholesterol-Free Diet in Two Cases. Warren M. Sperry, Ph.D., and Béla Schick, M.D., New York....1372

Abscess of the Liver of Umbilical Origin in Infants: Report of Two Cases. Tashio Kutsunai, M.D., Papaikou, Hawaii....1385

Intrapericardial Teratoma. Joseph Jellen, M.D., and Wilber E. Fisher, M.D., Rochester, N. Y....1397

Progress in Pediatrics:

Dermatorrhexis with Dermatochalasis and Arthrochalasis (the So-Called Ehlers-Danlos Syndrome). F. Ronchese, M.D., Providence, R. I....1403

News and Comment....1415

Abstracts from Current Literature....1416

Society Transactions:

Philadelphia Pediatric Society....1472

Chicago Pediatric Society....1477

Book Reviews....1492

Directory of Pediatric Societies....1495

General Index....1501